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=> file hcaplus
COST IN U.S. DOLLARS                                    SINCE FILE      TOTAL
                                                           ENTRY    SESSION
FULL ESTIMATED COST                                         0.84       0.84

FILE 'HCAPLUS' ENTERED AT 09:08:26 ON 11 MAR 2008
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FILE COVERS 1907 - 11 Mar 2008  VOL 148 ISS 11
FILE LAST UPDATED: 10 Mar 2008  (20080310/ED)

New CAS Information Use Policies, enter HELP USAGETERMS for details.

 This file contains CAS Registry Numbers for easy and accurate
 substance identification.

=> s neotintima or restenosis or stent

             0 NEOTINTIMA
          9253 RESTENOSIS
          5656 STENT
L1       13099 NEOTINTIMA OR RESTENOSIS OR STENT

=> s (PPAR or (peroxisome proliferator-activated receptor))

         10685 PPAR
         20400 PEROXISOME
         13861 PROLIFERATOR
        551870 ACTIVATED
        742262 RECEPTOR
          8211 PEROXISOME PROLIFERATOR-ACTIVATED RECEPTOR
                 (PEROXISOME(W)PROLIFERATOR(W)ACTIVATED(W)RECEPTOR)
L2       12209 (PPAR OR (PEROXISOME PROLIFERATOR-ACTIVATED RECEPTOR))

=> s phosphate or monoacylglycerol or diacylglycerol or pyrophosphate or
glycerophosphate

        591159 PHOSPHATE
           992 MONOACYLGLYCEROL
         11173 DIACYLGLYCEROL
         41912 PYROPHOSPHATE
          9101 GLYCEROPHOSPHATE
L3      632628 PHOSPHATE OR MONOACYLGLYCEROL OR DIACYLGLYCEROL OR PYROPHOSPHATE
                 OR GLYCEROPHOSPHATE

=> s l1 and l2

L4         100 L1 AND L2
```

```
=> s l1 and l2 and l3

L5            1 L1 AND L2 AND L3

=> s l4 and (PY<2004 or AY<2004 or PRY<2004)

      23979567 PY<2004
       4765121 AY<2004
       4243738 PRY<2004
L6           56 L4 AND (PY<2004 OR AY<2004 OR PRY<2004)

=> s l5 and (PY<2004 or AY<2004 or PRY<2004)

      23979567 PY<2004
       4765121 AY<2004
       4243738 PRY<2004
L7            0 L5 AND (PY<2004 OR AY<2004 OR PRY<2004)

=> file stnguide

COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                       ENTRY    SESSION
FULL ESTIMATED COST                                     2.69       3.53

FILE 'STNGUIDE' ENTERED AT 09:08:37 ON 11 MAR 2008
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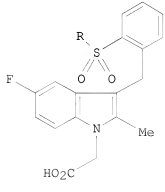
FILE CONTAINS CURRENT INFORMATION.
LAST RELOADED: Mar  7, 2008 (20080307/UP).

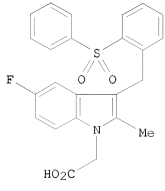
=> d l5 ti abs bib
YOU HAVE REQUESTED DATA FROM FILE 'HCAPLUS' - CONTINUE? (Y)/N:y



L5   ANSWER 1 OF 1  HCAPLUS  COPYRIGHT 2008 ACS on STN
TI   Preparation of indole compounds having CRTH2 antagonist activity for
     treating allergic diseases, asthma, and inflammatory conditions
GI
```



I



II

AB Compds. of general formula I (wherein R is Ph optionally substituted with one or more halo substituents) and their pharmaceutically acceptable salts, hydrates, solvates, complexes or prodrugs are antagonists at the CRTH2 receptor and are useful in the treatment of conditions mediated by PGD2 or other agonists binding to CRTH2. These include allergic diseases, asthmatic conditions and inflammatory diseases. A process for preparing I was addnl. claimed. Example compound II was prepared by reacting 2-(phenylsulfonyl)benzaldehyde with 2-(5-fluoro-2-methyl-1H-indol-1-yl)acetic acid and saponification of the resulting ester. In an assay measuring inhibition of 13,14-dihydro-15-keto-prostaglandin D2 induced blood eosinophilia in rats, II had an ED50 of 0.0025 μg/mL.

AN 2008:123834 HCAPLUS <<LOGINID::20080311>>

DN 148:183423

TI Preparation of indole compounds having CRTH2 antagonist activity for treating allergic diseases, asthma, and inflammatory conditions

IN Armer, Richard Edward; Wynne, Graham Michael

PA Oxagen Limited, UK

SO PCT Int. Appl., 68pp.
CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2008012511	A1	20080131	WO 2007-GB2761	20070720
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BH, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DO, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, ME, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW			
	RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, MT, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM			
PRAI	GB 2006-14608	A	20060722		
	GB 2006-24176	A	20061204		

RE.CNT 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> file hcaplus

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.24	9.43
DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-0.80

FILE 'HCAPLUS' ENTERED AT 09:10:57 ON 11 MAR 2008
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FILE COVERS 1907 - 11 Mar 2008 VOL 148 ISS 11
FILE LAST UPDATED: 10 Mar 2008 (20080310/ED)

New CAS Information Use Policies, enter HELP USAGETERMS for details.

This file contains CAS Registry Numbers for easy and accurate
substance identification.

```
=> s neointima or restenosis or stent

          1938 NEOINTIMA
          9253 RESTENOSIS
          5656 STENT
L8       14301 NEOINTIMA OR RESTENOSIS OR STENT

=> s (PPAR or (peroxisome proliferator-activated receptor))(4a)(inhibi? or block or
suppress)

         10685 PPAR
         20400 PEROXISOME
         13861 PROLIFERATOR
        551870 ACTIVATED
        742262 RECEPTOR
          8211 PEROXISOME PROLIFERATOR-ACTIVATED RECEPTOR
               (PEROXISOME(W)PROLIFERATOR(W)ACTIVATED(W)RECEPTOR)
       2016640 INHIBI?
        264158 BLOCK
         64386 SUPPRESS
L9        1527 (PPAR OR (PEROXISOME PROLIFERATOR-ACTIVATED RECEPTOR))(4A)(INHIB
               I? OR BLOCK OR SUPPRESS)

=> s l8 and l9

L10         29 L8 AND L9

=> s l10 and (PY<2004 or AY<2004 or PRY<2004)

      23979567 PY<2004
       4765121 AY<2004
       4243738 PRY<2004
L11         19 L10 AND (PY<2004 OR AY<2004 OR PRY<2004)

=> file stnguide

COST IN U.S. DOLLARS                          SINCE FILE      TOTAL
                                                 ENTRY    SESSION
FULL ESTIMATED COST                               2.69      12.12

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)    SINCE FILE      TOTAL
                                                 ENTRY    SESSION
CA SUBSCRIBER PRICE                               0.00      -0.80
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FILE 'STNGUIDE' ENTERED AT 09:11:04 ON 11 MAR 2008
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FILE CONTAINS CURRENT INFORMATION.
LAST RELOADED: Mar 7, 2008 (20080307/UP).

=> d l11 1-19 ti abs bib
YOU HAVE REQUESTED DATA FROM FILE 'HCAPLUS' - CONTINUE? (Y)/N:y

L11 ANSWER 1 OF 19 HCAPLUS COPYRIGHT 2008 ACS on STN
TI Lysophosphatidic acid analogs and inhibition of neointima formation
AB The phospholipid growth factor lysophosphatidic acids (LPAs) containing unsatd. fatty acids (18:1, 18:2 and 20:4) and fatty alcs. containing hydrocarbon chains with more than 4 carbons were capable of inducing a rapid formation of neointima, an initial step in the development of atherosclerotic plaque. LPAs with saturated fatty acids did not induce neointima formation. A Peroxisome Proliferator-Activated Receptors gamma (PPARγ)-specific agonist Rosiglitasone also induced a profound formation of neointima. GW9662, a selective and irreversible antagonist of PPARγ, abolished LPA- and Rosiglitazone-induced neointima formation, indicating that LPA-induced neointima formation requires the activation of PPARγ. These data suggest that LPA analogs that bind to but do not activate downstream signaling of PPARγ or antagonists of PPAR.gamma. that inhibit PPAR.gamma. signaling would be useful in the prevention and/or treatment of neointima formation and atherosclerosis.
AN 2004:857161 HCAPLUS <<LOGINID::20080311>>
DN 141:343506
TI Lysophosphatidic acid analogs and inhibition of neointima formation
IN Tigyi, Gabor; Baker, Daniel L.; Zhang, Chunxiang
PA USA
SO U.S. Pat. Appl. Publ., 23 pp.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2004204383	A1	20041014	US 2004-821739	20040409 <--
AU 2004229467	A1	20041028	AU 2004-229467	20040409 <--
AU 2004229467	B2	20070125		
CA 2521189	A1	20041028	CA 2004-2521189	20040409 <--
WO 2004091496	A2	20041028	WO 2004-US11016	20040409 <--
WO 2004091496	A3	20050324		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN,

TD, TG

EP 1613298 A2 20060111 EP 2004-759365 20040409 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK, HR

JP 2007525449 T 20070906 JP 2006-509874 20040409 <--

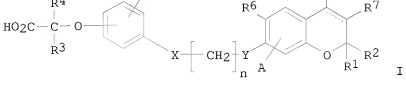
PRAI US 2003-462274P P 20030411 <--

WO 2004-US11016 W 20040409

L11 ANSWER 2 OF 19 HCAPLUS COPYRIGHT 2008 ACS on STN

TI PPARα-selective chromane and chromene compounds for the treatment of dyslipidemia and other lipid disorders, and preparation thereof

GI



I

AB A class of chromane and chromene compds. I [R1, R2, R4 = (un)substituted C1-3 alkyl; R3, R5, R7 = H, (un)substituted C1-3 alkyl; R6 = H, Cl, Me, CF3; A, B = H, Cl, F, Me, CF3; X, Y = O, S; n = 2, 3; dashed line = optional double bond], and pharmaceutically acceptable salts thereof, are useful as therapeutic compds., particularly in the treatment and control of hyperlipidemia, hypercholesterolemia, dyslipidemia, and other lipid disorders, and in delaying the onset of or reducing the risk of conditions and sequelae that are associated with these diseases, such as atherosclerosis. Compound preparation is included.

AN 2004:100986 HCAPLUS <<LOGINID::20080311>>

DN 140:157460

TI PPARα-selective chromane and chromene compounds for the treatment of dyslipidemia and other lipid disorders, and preparation thereof

IN Desai, Ranjit C.; Sahoo, Soumya

PA Merck & Co., Inc., USA

SO PCT Int. Appl., 57 pp.

CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2004010992	A1	20040205	WO 2003-US23499	20030725 <--
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
CA 2493913	A1	20040205	CA 2003-2493913	20030725 <--

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     AU 2003256911        A1    20040216    AU 2003-256911         20030725 <--
     EP 1539137           A1    20050615    EP 2003-771947         20030725 <--
        R:  AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
            IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK
     JP 2005538109        T     20051215    JP 2004-524924         20030725 <--
     US 2006089404        A1    20060427    US 2005-522646         20050926 <--
     US 7297715           B2    20071120
PRAI US 2002-399518P      P     20020730  <--
     WO 2003-US23499      W     20030725  <--
OS   MARPAT 140:157460
RE.CNT  2     THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD
              ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11  ANSWER 3 OF 19  HCAPLUS  COPYRIGHT 2008 ACS on STN
TI   Composition based on substituted 1,3-diphenylprop-en-1-one derivatives,
     preparation and use as PPARα agonists, antioxidants as well as
     antiinflammatory agents
GI


* STRUCTURE DIAGRAM TOO LARGE FOR DISPLAY -  AVAILABLE VIA OFFLINE PRINT *

AB   Title compds. I [wherein X1 = halo, R1, G1R1; X2 = H, thionitroso, OH,
     alkylcarbonyloxy, alkyloxy, SH, alkylthio, alkylcarbonylthio or X2 = O or
     S that forms a 2-phenyl-4H-1-benzopyran-4-one with the carbon-3 of the
     propene chain; X3 = R3, G3R3; X4 = halo, thionitroso, R4, G4R4; X5 = R5,
     G5R5; X6 = O, NH and derivs.; R1, R3, R4, R5 = independently H,
     (un)substituted alkyl; G1, G3, G4, G5 = independently O or S; with at
     least one of X1, X3, X4, or X5 of formula GR and one of the R1, R3, R4, or
     R5 is a substituted radical, and that radical form a cycle, or is associated
     with a group G; their optical and geometrical isomers, racemates,
     tautomers, salts, hydrates and mixts.; with the exclusion of certain
     compds.] were prepared as peroxisome proliferator-activated
     receptors-α (PPARα) agonists and as antioxidants for treating
     cerebral ischemia and related diseases.  For example, II was prepared by
     mixed-Aldol condensation of ketone III with 4-hydroxy-3,5-
     ditertbutylbenzaldehyde in the presence of ethanol/HCl.  In an antioxidant
     test, selected I (10-3 M) diminished the formation of oxidation product of
     LDL by AAPH by 33%.  Selected I were PPARα agonists, showing induced
     luciferase activity via PPARα/Gal4 transactivation with a factor of
     induction ranging from 10 to 60, 5-50 and 3-35 at 100 μM, 30 μM, and
     10 μM resp.  I and their compns. are useful for treating cardiovascular
     diseases, syndrome X, restenosis, diabetes, obesity,
     hypertension, inflammatory diseases, cancers or neoplasms (benign or
     malignant tumors), neurodegenerative diseases, dermatol. and the disorders
     related to the oxydative stress, for preventing and treating aging, and in
     particular cutaneous aging.
AN   2004:19750  HCAPLUS <<LOGINID::20080311>>
DN   140:76896
TI   Composition based on substituted 1,3-diphenylprop-en-1-one derivatives,
     preparation and use as PPARα agonists, antioxidants as well as
     antiinflammatory agents
IN   Najib, Jamila; Caumont Bertrand, Karine
PA   Genfit S.A., Fr.
SO   Fr. Demande, 66 pp.
     CODEN: FRXXBL
DT   Patent
LA   French
FAN.CNT 1
```

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	FR 2841784	A1	20040109	FR 2002-8570	20020708 <--
	FR 2841784	B1	20070302		
	CA 2490993	A1	20040115	CA 2003-2490993	20030708 <--
	WO 2004005243	A2	20040115	WO 2003-FR2128	20030708 <--
	WO 2004005243	A3	20040422		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	AU 2003264699	A1	20040123	AU 2003-264699	20030708 <--
	EP 1519908	A2	20050406	EP 2003-762750	20030708 <--
	EP 1519908	B1	20070613		
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
	BR 2003012399	A	20050412	BR 2003-12399	20030708 <--
	CN 1688532	A	20051026	CN 2003-816351	20030708 <--
	JP 2005532386	T	20051027	JP 2004-518891	20030708 <--
	AT 364588	T	20070715	AT 2003-762750	20030708 <--
	NZ 538052	A	20070928	NZ 2003-538052	20030708 <--
	ES 2287529	T3	20071216	ES 2003-762750	20030708 <--
	NO 2004005082	A	20041227	NO 2004-5082	20041122 <--
	MX 2005PA00425	A	20050722	MX 2005-PA425	20050107 <--
	US 2005171149	A1	20050804	US 2005-520078	20050404 <--
PRAI	FR 2002-8570	A	20020708 <--		
	WO 2003-FR2128	W	20030708 <--		

OS MARPAT 140:76896

RE.CNT 29 THERE ARE 29 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 4 OF 19 HCAPLUS COPYRIGHT 2008 ACS on STN

TI PPARγ ligands induce prostaglandin production in vascular smooth muscle cells: indomethacin acts as a peroxisome proliferator-activated receptor-γ antagonist

AB Peroxisome proliferator-activated receptor (PPAR)γ and inducible cyclooxygenase-2 (COX-2) are expressed in atherosclerotic lesions, particularly in the intimal monocytic and vascular smooth muscle cells. We have therefore studied the interaction between PPARγ and inducible cyclo-oxygenase (COX-2) in rat aortic vascular smooth muscle cells (RASMC)s. The synthetic PPARγ ligand rosiglitazone induced prostaglandin (PG) release from RASMCs, including that of PGD2, the precursor of the putative endogenous PPARγ ligand 15-deoxy-Δ12,14-prostaglandin J2. Moreover, rosiglitazone both synergized with IL-1β to further induce prostaglandin release and affected the expression of phospholipase A2 and COX-2. Rosiglitazone-induced prostaglandin release was inhibited by the PPAR.gamma. partial agonist GW0072 and the PPARγ antagonist GW9662. Rosiglitazone also induced RASMC apoptosis, an effect not explained as an autocrine effect of the induced-prostanoids, but on arachidonic acid release, as cell death was unaffected by either the nonselective COX inhibitor piroxicam or the selective COX-2 inhibitor DFP, but by inhibitors of either secretory or cytosolic phospholipase A2. In contrast, indomethacin, an alternative inhibitor of cyclooxygenase activity, inhibited both rosiglitazone-induced cell death, and

rosiglitazone-induced PPAR reporter gene activation.
AN 2003:826151 HCAPLUS <<LOGINID::20080311>>
DN 139:345691
TI PPARγ ligands induce prostaglandin production in vascular smooth muscle cells: indomethacin acts as a peroxisome proliferator-activated receptor-γ antagonist
AU Bishop-Bailey, David; Warner, Timothy D.
CS Dep. of Cardiac, Vascular and Inflammation Res., William Harvey Res. Inst., Barts and the London, Queen Mary's Sch. of Med. and Dentistry, London, EC1M 6BQ, UK
SO FASEB Journal (2003), 17(13), 1925-1927, 10.1096/fj.02-1075fje
CODEN: FAJOEC; ISSN: 0892-6638
PB Federation of American Societies for Experimental Biology
DT Journal
LA English
RE.CNT 29 THERE ARE 29 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 5 OF 19 HCAPLUS COPYRIGHT 2008 ACS on STN
TI Use of PPAR alpha agonists for the treatment of vascular and renal diseases
AB Activation of peroxisome proliferator activated receptor alpha (PPARα) by administration of therapeutic amts. of a PPAR α agonist, WY-14643, inhibits the proliferation of vascular smooth muscle cells, hepatoma cells and human renal proximal tubule cells. WY-14643 may be applicable as a medicament for the treatment of proliferative vascular disease (atherosclerosis, hypertension), revascularization-induced injury (restenosis) and chronic renal failure.
AN 2003:737571 HCAPLUS <<LOGINID::20080311>>
DN 139:255357
TI Use of PPAR alpha agonists for the treatment of vascular and renal diseases
IN Zahradka, Peter; Taylor, Carla
PA Can.
SO PCT Int. Appl., 33 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2003075911	A1	20030918	WO 2003-CA335	20030311 <--
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	CA 2481371	A1	20030918	CA 2003-2481371	20030311 <--
	AU 2003208238	A1	20030922	AU 2003-208238	20030311 <--
	US 2006052457	A1	20060309	US 2005-507495	20050817 <--
PRAI	US 2002-362243P	P	20020311 <--		
	WO 2003-CA335	W	20030311 <--		

RE.CNT 14 THERE ARE 14 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 6 OF 19 HCAPLUS COPYRIGHT 2008 ACS on STN
TI Inhibitory Activity of Clinical Thiazolidinedione Peroxisome Proliferator Activating Receptor-γ Ligands Toward Internal Mammary Artery, Radial Artery, and Saphenous Vein Smooth Muscle Cell Proliferation
AB Background- The proliferation of vascular smooth muscle cells (VSMCs) is a known response to arterial injury that is an important part of the process of restenosis and atherosclerosis. People with diabetes have an increased risk of cardiovascular disease resulting from accelerated coronary atherosclerosis. The newest drugs for Type 2 diabetes are thiazolidinediones, which are insulin-sensitizing peroxisome proliferator activating receptor-γ (PPARγ) ligands. We investigated the antiproliferative effects of troglitazone, rosiglitazone, and pioglitazone on VSMCs derived from the three vascular beds used for coronary artery bypass grafting: the internal mammary and radial artery and saphenous veins. Methods and Results- The three vessels yielded proliferating cells of slightly differing morphol. Inhibition of cell proliferation was assessed by cell counting and cell cycle studies by Western blotting for phosphorylated retinoblastoma protein. All three thiazolidinediones showed inhibitory potency toward cell proliferation with a potency troglitazone>rosiglitazone≈pioglitazone, and this potency profile was maintained toward the growth factor and insulin-stimulated phosphorylation of the retinoblastoma protein, which controls cell cycle progression. Conclusion- The inhibitory potency of clin. thiazolidinediones toward different vascular sources is dependent on the individual thiazolidinedione and very little on the vascular source.
AN 2003:373269 HCAPLUS <<LOGINID::20080311>>
DN 140:12803
TI Inhibitory Activity of Clinical Thiazolidinedione Peroxisome Proliferator Activating Receptor-γ Ligands Toward Internal Mammary Artery, Radial Artery, and Saphenous Vein Smooth Muscle Cell Proliferation
AU de Dios, Stephanie T.; Bruemmer, Dennis; Dilley, Rodney J.; Ivey, Melanie E.; Jennings, Garry L. R.; Law, Ronald E.; Little, Peter J.
CS Baker Heart Research Institute, Monash University, Melbourne, Australia
SO Circulation (2003), 107(20), 2548-2550
CODEN: CIRCAZ; ISSN: 0009-7322
PB Lippincott Williams & Wilkins
DT Journal
LA English
RE.CNT 16 THERE ARE 16 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 7 OF 19 HCAPLUS COPYRIGHT 2008 ACS on STN
TI PPAR.alpha. inhibits TGF-β induced β5 integrin transcription in vascular smooth muscle cells by interacting with Smad4
AB Integrins play an important role in vascular smooth muscle cell (VSMC) migration, a crucial event in the development of restenosis and atherosclerosis. Transforming growth factor-β (TGF-β) is highly expressed in restenotic and atherosclerotic lesions, and known to induce integrin expression. Peroxisome proliferator-activated receptor α (PPARα), a member of the nuclear receptor superfamily, regulates gene expression in a variety of vascular cells. The authors investigated the effects of PPARα ligands on TGF-β-induced β3 and β5 integrin expression and potential interaction between PPARα and TGF-β signaling. PPARα ligands WY-14643 (100 μM) and 5,8,11,14-eicosatetranoic acid (ETYA, 50 μM) inhibited TGF-β-induced β5 integrin protein expression by 72±6.8% and 73±7.1%, resp. (both P<0.05). TGF-β-stimulated β3 integrin expression was not affected by PPARα ligands. Both PPARα ligands also suppressed TGF-β-induced β5 integrin mRNA levels. PPAR.alpha. ligands inhibited TGF-β-inducible

transcription of β5 integrin by an interaction with a TGF-β response element between nucleotides -63 and -44, which contains a Sp1/Sp3 transcription factor binding site. Nuclear complexes binding to the TGF-β response region contained Sp1/Sp3 and TGF-β-regulated Smad 2, 3, and 4 transcription factors. TGF-β-stimulated Sp1/Smad4 nuclear complex formation was inhibited by WY-14643 and ETYA with a parallel induction of PPARα/Smad4 interactions. However, in vitro pull-down expts. failed to demonstrate direct binding between PPARα/Smad4. Both PPARα ligands blocked PDGF-directed migration of TGF-β-pretreated VSMCs, a process mediated, in part, by β5 integrins. The present study demonstrates that PPAR α activators inhibit TGF-β-induced β5 integrin transcription in VSMCs through a novel indirect interaction between ligand-activated PPARα and the TGF-β-regulated Smad4 transcription factors.

AN 2002:877961 HCAPLUS <<LOGINID::20080311>>
DN 138:199151
TI PPAR.alpha. inhibits TGF-β induced β5 integrin transcription in vascular smooth muscle cells by interacting with Smad4
AU Kintscher, Ulrich; Lyon, Christopher; Wakino, Shu; Bruemmer, Dennis; Feng, Xu; Goetze, Stephan; Graf, Kristof; Moustakas, Aristidis; Staels, Bart; Fleck, Eckart; Hsueh, Willa A.; Law, Ronald E.
CS School of Medicine, Division of Endocrinology, , Diabetes and Hypertension, Department of Medicine, University of California, Los Angeles, CA, USA
SO Circulation Research (2002), 91(11), e35-e44
CODEN: CIRUAL; ISSN: 0009-7330
PB Lippincott Williams & Wilkins
DT Journal
LA English
RE.CNT 43 THERE ARE 43 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 8 OF 19 HCAPLUS COPYRIGHT 2008 ACS on STN
TI Acyl sulfamides for treatment of obesity, diabetes and lipid disorders
AB A class of acyl sulfamides comprises compds. that are potent ligands for PPARγ receptors and generally have antagonist or partial agonist activity. The compds. may be useful in the treatment, control or prevention of obesity, non-insulin dependent diabetes mellitus (NIDDM), hyperglycemia, dyslipidemia, hyperlipidemia, hypercholesterolemia, hypertriglyceridemia, atherosclerosis, vascular restenosis, inflammation, and other PPARγ receptor-mediated diseases, disorders and conditions, alone or in combination with one or more other compds. Other compds. are selected from insulin sensitizers, insulin or insulin mimetics, sulfonylureas, α-glucosidase inhibitors, cholesterol lowering agents, PPAR.delta. agonists, antiobesity compds., an ileal bile acid transporter inhibitor, and agents intended for use in inflammatory conditions such as aspirin, nonsteroidal anti-inflammatory drugs, glucocorticoids, azulfidine, and cyclooxygenase-2 selective inhibitors.
AN 2002:594636 HCAPLUS <<LOGINID::20080311>>
DN 137:135097
TI Acyl sulfamides for treatment of obesity, diabetes and lipid disorders
IN Jones, A. Brian; Acton, John J., III
PA Merck & Co., Inc., USA
SO PCT Int. Appl., 64 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2002060388	A2	20020808	WO 2002-US3119	20020125 <--
	WO 2002060388	A3	20030227		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	CA 2434491	A1	20020808	CA 2002-2434491	20020125 <--
	AU 2002240235	A1	20020812	AU 2002-240235	20020125 <--
	EP 1357908	A2	20031105	EP 2002-706128	20020125 <--
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
	JP 2004521119	T	20040715	JP 2002-560584	20020125 <--
	US 2004073037	A1	20040415	US 2003-470483	20030729 <--
	US 6852738	B2	20050208		
PRAI	US 2001-264955P	P	20010130	<--	
	WO 2002-US3119	W	20020125	<--	

OS MARPAT 137:135097

L11 ANSWER 9 OF 19 HCAPLUS COPYRIGHT 2008 ACS on STN

TI Methods for treating inflammatory diseases using PPAR agonists

AB The present invention describes methods for the use of PPAR ligands in the treatment inflammatory endocrine, dermatol., cardiovascular immunol., neurol., ophthalmic, neoplastic, pulmonary diseases, and age-related dysregulations. In addition, methods are provided for treating said conditions and diseases comprising the step of administering to a human or an animal in need thereof a therapeutic amount of pharmacol. compns. comprising a pharmaceutically acceptable carrier, and a PPARγ agonist which cross-activates PPARα or PPARδ or both, or a PPARγ partial agonist, or a PPARγ/RXR agonist, effective to reverse, slow, stop, or prevent the pathol. inflammatory or degenerative process.

AN 2002:142506 HCAPLUS <<LOGINID::20080311>>

DN 136:177977

TI Methods for treating inflammatory diseases using PPAR agonists

IN Pershadsingh, Harrihar A.

PA USA

SO PCT Int. Appl., 42 pp.
CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 2

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2002013812	A1	20020221	WO 2001-US25668	20010816 <--
	W: AU, CA, MX, NZ, US				
	RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR				
	AU 2001088271	A5	20020225	AU 2001-88271	20010816 <--
PRAI	US 2000-225907P	P	20000817	<--	
	US 2000-230509P	P	20000906	<--	
	WO 2001-US25668	W	20010816	<--	

RE.CNT 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 10 OF 19 HCAPLUS COPYRIGHT 2008 ACS on STN
TI Peroxisome proliferator-activated receptor γ inhibits transforming growth factor β-induced connective tissue growth factor expression in human aortic smooth muscle cells by interfering with Smad3
AB Activation of peroxisome proliferator-activated receptor γ (PPARγ) after balloon injury significantly inhibits VSMC proliferation and neointima formation. However, the precise mechanisms of this inhibition have not been determined The authors hypothesized that activation of PPARγ in vascular injury could attenuate VSMC growth and matrix production during vascular lesion formation. Since connective tissue growth factor (CTGF) is a key factor regulating extracellular matrix production, abrogation of transforming growth factor β (TGF-β)-induced CTGF production by PPARγ activation may be one of the mechanisms through which PPAR.gamma. agonists inhibit neointima formation after vascular injury. In this study, the authors demonstrate that the PPARγ natural ligand (15-deoxyprostaglandin J2) and a synthetic ligand (GW7845) significantly inhibit TGF-β-induced CTGF production in a dose-dependent manner in HASMCs. In addition, suppression of CTGF mRNA expression is relieved by pretreatment with an antagonist of PPARγ (GW9662), suggesting that the inhibition of CTGF expression is mediated by PPARγ. To elucidate further the mol. mechanism by which PPAR.gamma. inhibits CTGF expression, an .apprx.2-kilobase pair CTGF promoter was cloned. The authors found that PPAR.gamma. activation inhibits TGF-β-induced CTGF promoter activity in a dose-dependent manner, and suppression of CTGF promoter activity by PPARγ activation is completely rescued by overexpression of Smad3, but not by Smad4. Furthermore, PPARγ phys. interacts with Smad3 but not Smad4 in vitro in glutathione S-transferase pull-down expts. Taken together, the data suggest that PPAR.gamma. inhibits TGF-β-induced CTGF expression in HASMCs by directly interfering with the Smad3 signaling pathway.
AN 2001:908512 HCAPLUS <<LOGINID::20080311>>
DN 136:198017
TI Peroxisome proliferator-activated receptor γ inhibits transforming growth factor β-induced connective tissue growth factor expression in human aortic smooth muscle cells by interfering with Smad3
AU Fu, Mingui; Zhang, Jifeng; Zhu, Xiaojun; Myles, David E.; Willson, Timothy M.; Liu, Xuedong; Chen, Yuqing E.
CS Cardiovascular Research Institute, Morehouse School of Medicine, Atlanta, GA, 30310, USA
SO Journal of Biological Chemistry (2001), 276(49), 45888-45894
CODEN: JBCHA3; ISSN: 0021-9258
PB American Society for Biochemistry and Molecular Biology
DT Journal
LA English
RE.CNT 30 THERE ARE 30 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 11 OF 19 HCAPLUS COPYRIGHT 2008 ACS on STN
TI Peroxisome proliferator-activated receptor-γ ligands inhibit nuclear but not cytosolic extracellular signal-regulated kinase/mitogen-activated protein kinase-regulated steps in vascular smooth muscle cell migration
AB Vascular smooth muscle cell (VSMC) migration involves adhesion, locomotion, and invasion regulated by various signaling mols., among which the extracellular signal-regulated kinase (ERK)/mitogen-activated protein kinases (MAPK) play a critical role. We have shown that the peroxisome proliferator-activated receptor-γ (PPAR-γ) ligands

troglitazone and rosiglitazone inhibit VSMC migration downstream of ERK MAPK. The purpose of the current study was to more specifically determine which step(s) in VSMC migration are targeted by inhibition of the ERK MAPK pathway or activation of PPAR-γ. VSMC adhesion was not affected by the ERK MAPK pathway inhibitor PD98059 or PPAR-γ ligands. Phosphorylation and activation of myosin light chain kinase (MLCK) play important roles in cell locomotion. Platelet-derived growth factor (PDGF)-induced MLCK phosphorylation (1.7-fold) was completely blocked by PD98059 at 30 μM ($p < 0.05$), but not by troglitazone or rosiglitazone. PDGF-directed migration (5.8-fold) was inhibited by PD98059 (-88% at 30 μM) and the MLCK inhibitor ML9 (0.1-1 μM, -84% at 1 μM) (all $p < 0.05$). The transcription factor Ets-1 mediates matrix metalloproteinase induction required for tissue invasion by VSMC. PDGF (20 ng/mL) stimulated an Ets-1 protein expression (14-fold at 60 min) in VSMC, which was inhibited by PD98059 (-72% at 30 μM), troglitazone (-69% at 20 μM), and rosiglitazone (-54% at 10 μM) (all $p < 0.05$). Immunohistochem. of rat aortae 2 h after balloon injury showed a dramatic upregulation of Ets-1, which was markedly inhibited in animals that had received troglitazone treatment. In contrast, phosphorylated ERK MAPK was not affected by troglitazone. These data are consistent with PPAR-γ ligands exerting their anti-migratory effects downstream of ERK MAPK activation by blocking nuclear events, such as Ets-1 expression, required for cell invasion in response to arterial injury.

AN 2001:887435 HCAPLUS <<LOGINID::20080311>>

DN 136:161114

TI Peroxisome proliferator-activated receptor-γ ligands inhibit nuclear but not cytosolic extracellular signal-regulated kinase/mitogen-activated protein kinase-regulated steps in vascular smooth muscle cell migration

AU Goetze, Stephan; Kintscher, Ulrich; Kim, Sarah; Meehan, Woerner P.; Kaneshiro, Kristina; Collins, Alan R.; Fleck, Eckart; Hsueh, Willa A.; Law, Ronald E.

CS Department of Medicine/Cardiology, Virchow Klinikum, Humboldt University Berlin and German Heart Institute Berlin, Berlin, 13353, Germany

SO Journal of Cardiovascular Pharmacology (2001), 38(6), 909-921
CODEN: JCPCDT; ISSN: 0160-2446

PB Lippincott Williams & Wilkins

DT Journal

LA English

RE.CNT 44 THERE ARE 44 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 12 OF 19 HCAPLUS COPYRIGHT 2008 ACS on STN

TI Control of vascular cell proliferation and migration by PPAR-γ: A new approach to the macrovascular complications of diabetes

AB A review with 82 refs. Compared with nondiabetic subjects, type 2 diabetic individuals are at an increased risk for coronary artery disease and coronary restenosis after angioplasty or stenting. Increased proliferation and migration of vascular smooth muscle cells (VSMCs) contribute importantly to the formation of both atherosclerotic and restenotic lesions. Therefore, pharmaceutical interventions targeting proteins that regulate VSMC growth or movement are a promising new approach to treat diabetes-associated cardiovascular disease. Peroxisome proliferator-activated receptor-γ (PPAR-γ) is a member of the nuclear receptor superfamily that, when activated by thiazolidinedione (TZD) insulin sensitizers, regulates a host of target genes. All of the major cells in the vasculature express PPAR-γ, including endothelial cells, VSMCs, and monocytes/macrophages. PPAR-γ is present in intimal macrophages and VSMCs in early human atheromas. In an animal model of vascular injury, PPAR-γ levels are substantially elevated in the neointima that forms after mech. injury of the

endothelium. Recent exptl. studies provide evidence that PPAR-γ may function to protect the vasculature from injury. Cell culture studies have shown that TZD PPAR-γ ligands inhibit both the proliferation and migration of VSMCs. These antiatherogenic activities of PPAR-γ may also occur in vivo, because TZDs inhibit lesion formation in several animal models. PPAR-γ ligands may also protect the vasculature indirectly by normalizing metabolic abnormalities of the diabetic milieu that increase cardiovascular risk. Activation of PPAR-γ, newly defined in vascular cells, may be a useful approach to protect the vasculature in diabetes.

AN 2001:136312 HCAPLUS <<LOGINID::20080311>>
DN 134:235155
TI Control of vascular cell proliferation and migration by PPAR-γ: A new approach to the macrovascular complications of diabetes
AU Hsueh, Willa A.; Jackson, Simon; Law, Ronald E.
CS Department of Medicine, the Endocrinology, Diabetes, and Hypertension Division, University of California School of Medicine, Los Angeles, CA, 90095-7073, USA
SO Diabetes Care (2001), 24(2), 392-397
CODEN: DICAD2; ISSN: 0149-5992
PB American Diabetes Association, Inc.
DT Journal; General Review
LA English
RE.CNT 82 THERE ARE 82 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 13 OF 19 HCAPLUS COPYRIGHT 2008 ACS on STN
TI Methods using PPAR.delta. inhibitors for treatment of vascular diseases, cancer, Alzheimer's disease, and inflammatory disorders, and drug screening methods
AB A method of preventing or reducing foam cell development from macrophages, or removing foam cells, in a patient comprises administering an effective amount of an inhibitor of PPAR.delta. activity. A method of preventing or treating a vascular disease associated with plaque formation and/or thrombotic blockage of the blood vessels in a patient comprises administering to the patient an effective amount of an inhibitor of PPAR.delta. activity. Also disclosed are methods for the treatment of cancer, Alzheimer's disease, and inflammatory disorders.
AN 2001:78255 HCAPLUS <<LOGINID::20080311>>
DN 134:141771
TI Methods using PPAR.delta. inhibitors for treatment of vascular diseases, cancer, Alzheimer's disease, and inflammatory disorders, and drug screening methods
IN Palmer, Colin Neil Alexander; Vosper, Helen; Wolf, Charles Roland
PA The University of Dundee, UK
SO PCT Int. Appl., 52 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2001007066	A2	20010201	WO 2000-EP6986	20000719 <--
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY,				

DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

CA	2378462	A1	20010201	CA 2000-2378462	20000719 <--
BR	2000012661	A	20020409	BR 2000-12661	20000719 <--
EP	1200114	A2	20020502	EP 2000-956238	20000719 <--

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL

TR	200200211	T2	20020621	TR 2002-211	20000719 <--
HU	2002001966	A2	20020928	HU 2002-1966	20000719 <--
HU	2002001966	A3	20050128		
JP	2003505058	T	20030212	JP 2001-511949	20000719 <--
TR	200501763	T2	20050822	TR 2005-1763	20000719 <--
IN	2001MN01670	A	20050304	IN 2001-MN1670	20011231 <--
NO	2002000326	A	20020320	NO 2002-326	20020122 <--
ZA	2002000542	A	20030415	ZA 2002-542	20020122 <--
MX	2002PA00880	A	20030714	MX 2002-PA880	20020123 <--
AU	2004212557	A1	20041014	AU 2004-212557	20040916 <--
IN	2008MN00046	A	20080222	IN 2008-MN46	20080108 <--
PRAI	GB 1999-17405	A	19990723	<--	
	AU 2000-68259	A3	20000710	<--	
	WO 2000-EP6986	W	20000719	<--	
	IN 2001-MN1670	A3	20011231	<--	

L11 ANSWER 14 OF 19 HCAPLUS COPYRIGHT 2008 ACS on STN

TI PPARs and atherosclerosis

AB A review, with 48 refs. PPARs are key players in lipid and glucose metabolism, which have been implicated in metabolic diseases, such as dyslipidemia and diabetes, predisposing to atherosclerosis. Whereas PPARγ promotes lipid storage via its effects on adipocyte differentiation and function, PPARα stimulates the β-oxidative degradation of fatty acids. PPARα-deficient mice exhibit a prolonged response to inflammatory stimuli suggesting that PPARα could be a mediator of inflammatory control. Fibrates, synthetic PPARα ligands, decrease atherosclerotic lesion progression, even in the absence of atherogenic lipoprotein lowering suggesting a function of PPARs at the vascular wall. Therefore, the expression and function of PPARs in human vascular smooth muscle cells (SMC), macrophages and endothelial cells (EC) was analyzed. Whereas human aortic SMC and coronary EC express mainly PPARα, differentiated macrophages express both PPARα and PPARγ. In SMC and EC PPAR.alpha. activators resp. inhibit interleukin (IL)1-induced IL-6 and prostaglandin (PG) production and thrombin-induced endothelin-1 production In differentiated macrophages, activation of PPARγ results in apoptosis induction, as measured by the TUNEL assay and the appearance of the active proteolytic subunits of the cell death protease caspase-3. In all cell types PPARs act by neg. interfering with the NFκB and AP-1 signaling pathways. These data indicate a novel function for PPARs in cells of the vascular wall in modulating vasomotricity, inflammatory response and cell proliferation with likely consequences in atherosclerosis and restenosis.

AN 2000:647715 HCAPLUS <<LOGINID::20080311>>

DN 134:129171

TI PPARs and atherosclerosis

AU Torra, InEs Pineda; Fruchart, Jean-Charles; Staels, Bart

CS INSERM U.325, Dep. d'AthErosclErose, Institut Pasteur de Lille, Lille, 59019, Fr.

SO Lipoprotein Metabolism and Atherogenesis, [International Symposium on Lipoprotein Metabolism and Atherogenesis], Kyoto, Japan, Dec. 5-8, 1998 (2000), Meeting Date 1998, 88-95. Editor(s): Kita, Toru; Yokode, Masayuki. Publisher: Springer-Verlag Tokyo, Tokyo, Japan.
CODEN: 69AIQ9

DT Conference; General Review
LA English
RE.CNT 48 THERE ARE 48 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 15 OF 19 HCAPLUS COPYRIGHT 2008 ACS on STN
TI Peroxisome proliferator-activated
receptor-γ ligands inhibit nitric oxide synthesis
in vascular smooth muscle cells
AB Peroxisome proliferator-activated receptor-γ (PPARγ) is a key
player in glucose metabolism If PPARγ ligands modulate nitric oxide
(NO) synthesis in the vascular tissue, they may affect the process of
plaque formation and postangioplasty restenosis. We
investigated the effects of PPARγ ligands on NO synthesis in
vascular smooth muscle cells. Incubation of cultures with
interleukin-1β (10 ng/mL) for 24 h caused a significant increase in
the production of nitrite, a stable metabolite of NO, in cultured rat vascular
smooth muscle cells. The PPARγ agonists troglitazone and
15-deoxy-Δ12,14-prostaglandin J2 (15d-PG J2) dose-dependently
inhibited nitrite production by interleukin-1β-stimulated vascular smooth
muscle cells. Decreased interleukin-1β-induced nitrite production by the
PPARγ agonist was accompanied by decreased inducible NO synthase
mRNA and protein accumulation. Interleukin-1β induced nuclear
factor-κB activation in vascular smooth muscle cells, and both
troglitazone and 15d-PG J2 markedly suppressed this nuclear
factor-κB activation. PPAR.gamma. ligands inhibit
NO synthesis in cytokine-stimulated vascular smooth muscle cells,
suggesting that these agonists may act directly on the vascular smooth
muscle and influence the process of atherosclerosis and restenosis
.
AN 2000:444444 HCAPLUS <<LOGINID::20080311>>
DN 133:305468
TI Peroxisome proliferator-activated
receptor-γ ligands inhibit nitric oxide synthesis
in vascular smooth muscle cells
AU Ikeda, Uichi; Shimpo, Masahisa; Murakami, Yoshiaki; Shimada, Kazuyuki
CS Department of Cardiology, Jichi Medical School, Tochigi, 329-0498, Japan
SO Hypertension (2000), 35(6), 1232-1236
CODEN: HPRTDN; ISSN: 0194-911X
PB Lippincott Williams & Wilkins
DT Journal
LA English
RE.CNT 45 THERE ARE 45 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 16 OF 19 HCAPLUS COPYRIGHT 2008 ACS on STN
TI Expression and function of PPARγ in rat and human vascular smooth
muscle cells
AB Peroxisome proliferator-activated receptor-γ (PPARγ) is
activated by fatty acids, eicosanoids, and insulin-sensitizing
thiazolidinediones (TZDs). The TZD troglitazone (TRO) inhibits vascular
smooth muscle cell (VSMC) proliferation and migration in vitro and in
post-injury intimal hyperplasia. Rat and human VSMCs expressed mRNA and
nuclear PPARγ1. Three PPARγ ligands, the TZDs TRO and
rosiglitazone and the prostanoid 15-deoxy-Δ12,14-prostaglandin J2
(15d-PGJ2), all inhibited VSMC proliferation and migration. PPARγ
was upregulated in rat neointima at 7 days and 14 days after
balloon injury and was also present in early human atheroma and precursor
lesions. Thus, pharmacol. activation of PPAR.gamma. expressed
in VSMCs inhibits their proliferation and migration, potentially
limiting restenosis and atherosclerosis. These receptors are

upregulated during vascular injury.
AN 2000:240919 HCAPLUS <<LOGINID::20080311>>
DN 133:148479
TI Expression and function of PPARγ in rat and human vascular smooth muscle cells
AU Law, Ronald E.; Goetze, Stephan; Xi, Xiao-Ping; Jackson, Simon; Kawano, Yasuko; Demer, Linda; Fishbein, Michael C.; Meehan, Woerner P.; Hsueh, Willa A.
CS Department of Medicine, University of California at Los Angeles School of Medicine, Los Angeles, CA, 90095, USA
SO Circulation (2000), 101(11), 1311-1318
CODEN: CIRCAZ; ISSN: 0009-7322
PB Lippincott Williams & Wilkins
DT Journal
LA English
RE.CNT 33 THERE ARE 33 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 17 OF 19 HCAPLUS COPYRIGHT 2008 ACS on STN
TI PPAR.gamma.-ligands inhibit migration mediated by multiple chemoattractants in vascular smooth muscle cells
AB The purpose of this study was to determine the effect of the peroxisome proliferator-activated receptor γ-(PPARγ) ligands troglitazone (TRO), rosiglitazone (RSG), and 15-deoxy-Δ prostaglandin J2 (15d-PGJ2) on vascular smooth muscle cell (VSMC) migration directed by multiple chemoattractants. Involvement of mitogen-activated protein kinase (MAPK) in migration also was examined, because TRO was previously shown to inhibit nuclear events stimulated by this pathway during mitogenic signaling in VSMCs. Migration of rat aortic VSMCs was induced 5.4-fold by PDGF, 4.6-fold by thrombin, and 2.3-fold by insulin-like growth factor I (IGF-I; all values of $p < 0.05$). The PPARγ ligands 15d-PGJ2, RSG, or TRO all inhibited VSMC migration with the following order of potency: 15d-PGJ2 > RSG > TRO. Inhibition of MAPK signaling with PD98059 completely blocked PDGF-, thrombin-, and IGF-I-induced migration. All chemoattractants induced MAPK activation. PPAR.gamma. ligands did not inhibit MAPK activation, suggesting a nuclear effect of these ligands downstream of MAPK. The importance of nuclear events was confirmed because actinomycin D also blocked migration. We conclude that PPAR.gamma. ligands are potent inhibitors of VSMC migration pathways, dependent on MAPK and nuclear events. PPARγ ligands act downstream of the cytoplasmic activation of MAPK and appear to exert their effects in the nucleus. Because VSMC migration plays an important role in the formation of atherosclerotic lesions and restenosis, PPARγ ligands like TRO and RSG, which ameliorate insulin resistance in humans, also may protect the vasculature from diabetes-enhanced injury.
AN 1999:275338 HCAPLUS <<LOGINID::20080311>>
DN 131:67939
TI PPAR.gamma.-ligands inhibit migration mediated by multiple chemoattractants in vascular smooth muscle cells
AU Goetze, Stephan; Xi, Xiao-Ping; Kawano, Hiroaki; Gotlibowski, Tina; Fleck, Eckart; Hsueh, Willa A.; Law, Ronald E.
CS School of Medicine, Division of Endocrinology, Diabetes and Hypertension, University of California, Los Angeles, Los Angeles, CA, 90095, USA
SO Journal of Cardiovascular Pharmacology (1999), 33(5), 798-806
CODEN: JCPCDT; ISSN: 0160-2446
PB Lippincott Williams & Wilkins
DT Journal
LA English
RE.CNT 56 THERE ARE 56 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 18 OF 19 HCAPLUS COPYRIGHT 2008 ACS on STN
TI Peroxisome proliferator-activated receptor gamma activators inhibit gene expression and migration in human vascular smooth muscle cells
AB Migration of vascular smooth muscle cells (VSMCs) plays an important role in atherogenesis and restenosis after arterial interventions. The expression of matrix metalloproteinases (MMPs), particularly MMP-9, contributes to VSMC migration. This process requires degradation of basal laminae and other components of the arterial extracellular matrix. Peroxisome proliferator-activated receptors (PPARs), members of the nuclear receptor family, regulate gene expression after activation by various ligands. Recent studies have suggested opposing effects of PPAR gamma (PPARγ) activation on atherogenesis. The present study tested the hypotheses that human VSMCs express PPAR alpha (PPARα) and PPARγ and that PPAR agonists in VSMCs modulate MMP-9 expression and activity, as well as VSMC migration. Human VSMCs expressed PPARα and PPARγ mRNA and protein. Treatment of VSMCs with the PPARγ ligands troglitazone and the naturally occurring 15-deoxy-Δ12,14-prostaglandin J2 (15d-PGJ2) decreased phorbol 12-myristate 13-acetate-induced MMP-9 mRNA and protein levels, as well as MMP-9 gelatinolytic activity in the supernatants in a concentration-dependent manner. Six different PPARα activators lacked such effects. Addition of prostaglandin F2α, known to limit PPARγ activity, diminished the MMP-9 inhibition seen with either troglitazone or 15d-PGJ2, further implicating PPARγ in these effects. Finally, troglitazone and 15d-PGJ2 inhibited the platelet-derived growth factor-BB-induced migration of VSMCs in vitro in a concentration-dependent manner. PPARγ activation may regulate VSMC migration and expression and activity of MMP-9. Thus, PPARγ activation in VSMCs, via the antidiabetic agent troglitazone or naturally occurring ligands, may act to counterbalance other potentially proatherosclerotic PPARγ effects.
AN 1998:798928 HCAPLUS <<LOGINID::20080311>>
DN 130:137272
TI Peroxisome proliferator-activated receptor gamma activators inhibit gene expression and migration in human vascular smooth muscle cells
AU Marx, Nikolaus; Schonbeck, Uwe; Lazar, Mitchell A.; Libby, Peter; Plutzky, Jorge
CS Vascular Medicine and Atherosclerosis Unit, Cardiovascular Division, Department of Medicine, Harvard Medical School, Brigham and Women's Hospital, Boston, MA, 02115, USA
SO Circulation Research (1998), 83(11), 1097-1103
CODEN: CIRUAL; ISSN: 0009-7330
PB Lippincott Williams & Wilkins
DT Journal
LA English
RE.CNT 34 THERE ARE 34 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 19 OF 19 HCAPLUS COPYRIGHT 2008 ACS on STN
TI Activation of human aortic smooth-muscle cells is inhibited by PPAR.alpha. but not by PPARγ activators
AB Peroxisome proliferator-activated receptors (PPARs) are key players in lipid and glucose metabolism and are implicated in metabolic disorders predisposing to atherosclerosis, such as dyslipidemia and diabetes. Whereas PPARγ promotes lipid storage by regulating adipocyte differentiation, PPARα stimulates the β-oxidative degradation of fatty acids. PPARα-deficient mice show a prolonged response to inflammatory stimuli, suggesting that PPARα is also a modulator of inflammation. Hypolipidemic fibrate drugs are PPAR.alpha.

ligands that inhibit the progressive formation of atherosclerotic lesions, which involves chronic inflammatory processes, even in the absence of their atherogenic lipoprotein-lowering effect. Here we show that PPARα is expressed in human aortic smooth-muscle cells, which participate in plaque formation and post-angioplasty re-stenosis. In these smooth-muscle cells, we find that PPARα ligands, and not PPAR.gamma. ligands, inhibit interleukin-1-induced production of interleukin-6 and prostaglandin and expression of cyclooxygenase-2. This inhibition of cyclooxygenase-2 induction occurs transcriptionally as a result of PPARα repression of NF-κB signalling. In hyperlipidemic patients, fenofibrate treatment decreases the plasma concns. of interleukin-6, fibrinogen and C-reactive protein. We conclude that activators of PPAR.alpha. inhibit the inflammatory response of aortic smooth-muscle cells and decrease the concentration of plasma acute-phase proteins, indicating that PPARα in the vascular wall may influence the process of atherosclerosis and re-stenosis.

AN 1998:439036 HCAPLUS <<LOGINID::20080311>>
DN 129:173485
TI Activation of human aortic smooth-muscle cells is inhibited by PPAR.alpha. but not by PPARγ activators
AU Staels, Bart; Koenig, Wolfgang; Habib, Aida; Merval, Regine; Lebret, Marilyne; Torra, Ines Pineda; Delerive, Philippe; Fadel, Abdessamad; Chinetti, Giulia; Fruchart, Jean-Charles; Najib, Jamila; Maclouf, Jacques; Tedgui, Alain
CS U325 INSERM, Dep. d'Atherosclerose, Inst. Pasteur, Lille, 59019, Fr.
SO Nature (London) (1998), 393(6687), 790-793
CODEN: NATUAS; ISSN: 0028-0836
PB Macmillan Magazines
DT Journal
LA English
RE.CNT 30 THERE ARE 30 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> d his

(FILE 'HOME' ENTERED AT 09:06:15 ON 11 MAR 2008)

FILE 'HCAPLUS' ENTERED AT 09:08:26 ON 11 MAR 2008
L1 13099 S NEOTINTIMA OR RESTENOSIS OR STENT
L2 12209 S (PPAR OR (PEROXISOME PROLIFERATOR-ACTIVATED RECEPTOR))
L3 632628 S PHOSPHATE OR MONOACYLGLYCEROL OR DIACYLGLYCEROL OR PYROPHOSPH
L4 100 S L1 AND L2
L5 1 S L1 AND L2 AND L3
L6 56 S L4 AND (PY<2004 OR AY<2004 OR PRY<2004)
L7 0 S L5 AND (PY<2004 OR AY<2004 OR PRY<2004)

FILE 'STNGUIDE' ENTERED AT 09:08:37 ON 11 MAR 2008

FILE 'HCAPLUS' ENTERED AT 09:08:51 ON 11 MAR 2008

FILE 'STNGUIDE' ENTERED AT 09:08:51 ON 11 MAR 2008

FILE 'HCAPLUS' ENTERED AT 09:10:57 ON 11 MAR 2008
L8 14301 S NEOINTIMA OR RESTENOSIS OR STENT
L9 1527 S (PPAR OR (PEROXISOME PROLIFERATOR-ACTIVATED RECEPTOR))(4A)(IN
L10 29 S L8 AND L9
L11 19 S L10 AND (PY<2004 OR AY<2004 OR PRY<2004)

FILE 'STNGUIDE' ENTERED AT 09:11:04 ON 11 MAR 2008

FILE 'HCAPLUS' ENTERED AT 09:11:13 ON 11 MAR 2008

FILE 'STNGUIDE' ENTERED AT 09:11:15 ON 11 MAR 2008

=> log hold

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.06	70.22

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-16.00

SESSION WILL BE HELD FOR 120 MINUTES
STN INTERNATIONAL SESSION SUSPENDED AT 09:11:22 ON 11 MAR 2008

Connecting via Winsock to STN

Welcome to STN International! Enter x:x

LOGINID:SSPTAEXO1623

PASSWORD:
* * * * * * RECONNECTED TO STN INTERNATIONAL * * * * * *
SESSION RESUMED IN FILE 'STNGUIDE' AT 09:40:51 ON 11 MAR 2008
FILE 'STNGUIDE' ENTERED AT 09:40:51 ON 11 MAR 2008
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COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.06	70.22

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-16.00

=> file hcaplus

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.12	70.28

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-16.00

FILE 'HCAPLUS' ENTERED AT 09:42:17 ON 11 MAR 2008
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FILE COVERS 1907 - 11 Mar 2008 VOL 148 ISS 11
FILE LAST UPDATED: 10 Mar 2008 (20080310/ED)

New CAS Information Use Policies, enter HELP USAGETERMS for details.

This file contains CAS Registry Numbers for easy and accurate substance identification.

=> s neointima or neointimal or stent

```
          1938 NEOINTIMA
          2352 NEOINTIMAL
          5656 STENT
L12       8522 NEOINTIMA OR NEOINTIMAL OR STENT
```

=> s l2 and l12

```
L13         45 L2 AND L12
```

=> s l9 and l12

```
L14         16 L9 AND L12
```

=> s l3 and (PY<2004 or AY<2004 or PRY<2004)

```
      23979567 PY<2004
       4765121 AY<2004
       4243738 PRY<2004
L15     550214 L3 AND (PY<2004 OR AY<2004 OR PRY<2004)
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=> s l3 and (PY<2004 or AY<2004 or PRY<2004)

```
      23979567 PY<2004
       4765121 AY<2004
       4243738 PRY<2004
L16     550214 L3 AND (PY<2004 OR AY<2004 OR PRY<2004)
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=> file stnguide

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	2.69	72.97

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-16.00

FILE 'STNGUIDE' ENTERED AT 09:42:28 ON 11 MAR 2008
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FILE CONTAINS CURRENT INFORMATION.
LAST RELOADED: Mar 7, 2008 (20080307/UP).

=> file hcaplus

COST IN U.S. DOLLARS	SINCE FILE	TOTAL

	ENTRY	SESSION
FULL ESTIMATED COST	0.06	73.03

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-16.00

FILE 'HCAPLUS' ENTERED AT 09:42:49 ON 11 MAR 2008
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
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FILE COVERS 1907 - 11 Mar 2008 VOL 148 ISS 11
FILE LAST UPDATED: 10 Mar 2008 (20080310/ED)

New CAS Information Use Policies, enter HELP USAGETERMS for details.

This file contains CAS Registry Numbers for easy and accurate substance identification.

=> s l13 and (PY<2004 or AY<2004 or PRY<2004)

```
      23979567 PY<2004
       4765121 AY<2004
       4243738 PRY<2004
L17         15 L13 AND (PY<2004 OR AY<2004 OR PRY<2004)
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=> s l14 and (PY<2004 or AY<2004 or PRY<2004)

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      23979567 PY<2004
       4765121 AY<2004
       4243738 PRY<2004
L18          6 L14 AND (PY<2004 OR AY<2004 OR PRY<2004)
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=> file stnguide

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	2.69	75.72

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-16.00

FILE 'STNGUIDE' ENTERED AT 09:42:55 ON 11 MAR 2008
USE IS SUBJECT TO THE TERMS OF YOUR CUSTOMER AGREEMENT

FILE CONTAINS CURRENT INFORMATION.
LAST RELOADED: Mar 7, 2008 (20080307/UP).

```
=> d l18 1-6 ti abs bib
YOU HAVE REQUESTED DATA FROM FILE 'HCAPLUS' - CONTINUE? (Y)/N:y



L18  ANSWER 1 OF 6  HCAPLUS  COPYRIGHT 2008 ACS on STN
TI   Lysophosphatidic acid analogs and inhibition of neointima
     formation
AB   The phospholipid growth factor lysophosphatidic acids (LPAs) containing
     unsatd. fatty acids (18:1, 18:2 and 20:4) and fatty alcs. containing
     hydrocarbon chains with more than 4 carbons were capable of inducing a
     rapid formation of neointima, an initial step in the development
     of atherosclerotic plaque.  LPAs with saturated fatty acids did not induce
     neointima formation.  A Peroxisome Proliferator-Activated
     Receptors gamma (PPARγ)-specific agonist Rosiglitasone also induced
     a profound formation of neointima.  GW9662, a selective and
     irreversible antagonist of PPARγ, abolished LPA- and
     Rosiglitazone-induced neointima formation, indicating that
     LPA-induced neointima formation requires the activation of
     PPARγ.  These data suggest that LPA analogs that bind to but do not
     activate downstream signaling of PPARγ or antagonists of
     PPAR.gamma. that inhibit PPAR.gamma. signaling
     would be useful in the prevention and/or treatment of neointima
     formation and atherosclerosis.
AN   2004:857161  HCAPLUS <<LOGINID::20080311>>
DN   141:343506
TI   Lysophosphatidic acid analogs and inhibition of neointima
     formation
IN   Tigyi, Gabor; Baker, Daniel L.; Zhang, Chunxiang
PA   USA
SO   U.S. Pat. Appl. Publ., 23 pp.
     CODEN: USXXCO
DT   Patent
LA   English
FAN.CNT 1
     PATENT NO.          KIND   DATE        APPLICATION NO.        DATE
     --------------      ----   --------    -------------------    --------
PI   US 2004204383       A1     20041014    US 2004-821739         20040409 <--
     AU 2004229467       A1     20041028    AU 2004-229467         20040409 <--
     AU 2004229467       B2     20070125
     CA 2521189          A1     20041028    CA 2004-2521189        20040409 <--
     WO 2004091496       A2     20041028    WO 2004-US11016        20040409 <--
     WO 2004091496       A3     20050324
         W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH,
             CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD,
             GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC,
             LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI,
             NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY,
             TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
         RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ,
             BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE,
             ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI,
             SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN,
             TD, TG
     EP 1613298          A2     20060111    EP 2004-759365         20040409 <--
         R:  AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
             IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK, HR
     JP 2007525449       T      20070906    JP 2006-509874         20040409 <--
PRAI US 2003-462274P     P      20030411  <--
```

WO 2004-US11016 W 20040409

L18 ANSWER 2 OF 6 HCAPLUS COPYRIGHT 2008 ACS on STN
TI PPAR.gamma. ligand inhibits osteopontin gene expression through interference with binding of nuclear factors to A/T-rich sequence in THP-1 cells
AB Peroxisome proliferator-activated receptor γ(PPARγ) is a member of the nuclear receptor superfamily that acts as a key player in adipocyte differentiation, glucose metabolism, and macrophage differentiation. Osteopontin (OPN) a component of extracellular matrix, is elevated during neointimal formation in the vessel wall and is synthesized by macrophages in atherosclerotic plaques. In the present study. we investigated the mol. mechanisms regulating OPN gene expression by PPARγ in THP-1 cells, a cell line derived from human monocytic leukemia cells. Northern and Western blot analyses showed that exposure of THP-1 cells to PMA (phorbol 12-myristate 13-acetate) increases OPN mRNA and protein levels in a time-dependent manner. PMA-induced OPN expression was significantly decreased by troglitazone (Tro) and other PPARγ ligands. Transient transfection assays of the human OPN promoter/luciferase construct showed that PPARγ represses OPN promoter activity, and the PPARγ-responsive region within the OPN promoter lies between -1000 and -970 relative to the transcription start site. Site-specific mutation anal. and electrophoretic mobility shift assays indicated that a homeobox-like A/T-rich sequence between -990 and 981, which functions as a binding site for PMA-induced nuclear factors other than PPARγ, mediates the repression of OPN expression by Tro. Furthermore, concatenated A/T-rich sequences conferred the PPARγ responsiveness on the heterologous promoter. Taken together, these data suggest that PPAR.gamma. ligand inhibits OPN gene expression through the interference with the binding of nuclear factors to A/T-rich sequence in THP-1 cells.
AN 2002:162012 HCAPLUS <<LOGINID::20080311>>
DN 136:338695
TI PPAR.gamma. ligand inhibits osteopontin gene expression through interference with binding of nuclear factors to A/T-rich sequence in THP-1 cells
AU Oyama, Yuko; Akuzawa, Nobuhiro; Nagai, Ryozo; Kurabayashi, Masahiko
CS Second Department of Internal Medicine, Gunma University School of Medicine, Maebashi, 371-8511, Japan
SO Circulation Research (2002), 90(3), 348-355
CODEN: CIRUAL; ISSN: 0009-7330
PB Lippincott Williams & Wilkins
DT Journal
LA English
RE.CNT 34 THERE ARE 34 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L18 ANSWER 3 OF 6 HCAPLUS COPYRIGHT 2008 ACS on STN
TI Peroxisome proliferator-activated receptor γ inhibits transforming growth factor β-induced connective tissue growth factor expression in human aortic smooth muscle cells by interfering with Smad3
AB Activation of peroxisome proliferator-activated receptor γ (PPARγ) after balloon injury significantly inhibits VSMC proliferation and neointima formation. However, the precise mechanisms of this inhibition have not been determined The authors hypothesized that activation of PPARγ in vascular injury could attenuate VSMC growth and matrix production during vascular lesion formation. Since connective tissue growth factor (CTGF) is a key factor regulating extracellular matrix production, abrogation of transforming growth factor β (TGF-β)-induced CTGF production by PPARγ activation may be

one of the mechanisms through which PPAR.gamma. agonists inhibit neointima formation after vascular injury. In this study, the authors demonstrate that the PPARγ natural ligand (15-deoxyprostaglandin J2) and a synthetic ligand (GW7845) significantly inhibit TGF-β-induced CTGF production in a dose-dependent manner in HASMCs. In addition, suppression of CTGF mRNA expression is relieved by pretreatment with an antagonist of PPARγ (GW9662), suggesting that the inhibition of CTGF expression is mediated by PPARγ. To elucidate further the mol. mechanism by which PPAR.gamma. inhibits CTGF expression, an .apprx.2-kilobase pair CTGF promoter was cloned. The authors found that PPAR.gamma. activation inhibits TGF-β-induced CTGF promoter activity in a dose-dependent manner, and suppression of CTGF promoter activity by PPARγ activation is completely rescued by overexpression of Smad3, but not by Smad4. Furthermore, PPARγ phys. interacts with Smad3 but not Smad4 in vitro in glutathione S-transferase pull-down expts. Taken together, the data suggest that PPAR.gamma. inhibits TGF-β-induced CTGF expression in HASMCs by directly interfering with the Smad3 signaling pathway.

AN 2001:908512 HCAPLUS <<LOGINID::20080311>>

DN 136:198017

TI Peroxisome proliferator-activated receptor γ inhibits transforming growth factor β-induced connective tissue growth factor expression in human aortic smooth muscle cells by interfering with Smad3

AU Fu, Mingui; Zhang, Jifeng; Zhu, Xiaojun; Myles, David E.; Willson, Timothy M.; Liu, Xuedong; Chen, Yuqing E.

CS Cardiovascular Research Institute, Morehouse School of Medicine, Atlanta, GA, 30310, USA

SO Journal of Biological Chemistry (2001), 276(49), 45888-45894
CODEN: JBCHA3; ISSN: 0021-9258

PB American Society for Biochemistry and Molecular Biology

DT Journal

LA English

RE.CNT 30 THERE ARE 30 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L18 ANSWER 4 OF 6 HCAPLUS COPYRIGHT 2008 ACS on STN

TI Control of vascular cell proliferation and migration by PPAR-γ: A new approach to the macrovascular complications of diabetes

AB A review with 82 refs. Compared with nondiabetic subjects, type 2 diabetic individuals are at an increased risk for coronary artery disease and coronary restenosis after angioplasty or stenting. Increased proliferation and migration of vascular smooth muscle cells (VSMCs) contribute importantly to the formation of both atherosclerotic and restenotic lesions. Therefore, pharmaceutical interventions targeting proteins that regulate VSMC growth or movement are a promising new approach to treat diabetes-associated cardiovascular disease. Peroxisome proliferator-activated receptor-γ (PPAR-γ) is a member of the nuclear receptor superfamily that, when activated by thiazolidinedione (TZD) insulin sensitizers, regulates a host of target genes. All of the major cells in the vasculature express PPAR-γ, including endothelial cells, VSMCs, and monocytes/macrophages. PPAR-γ is present in intimal macrophages and VSMCs in early human atheromas. In an animal model of vascular injury, PPAR-γ levels are substantially elevated in the neointima that forms after mech. injury of the endothelium. Recent exptl. studies provide evidence that PPAR-γ may function to protect the vasculature from injury. Cell culture studies have shown that TZD PPAR-γ ligands inhibit both the proliferation and migration of VSMCs. These antiatherogenic activities of PPAR-γ may also occur in vivo, because TZDs inhibit

lesion formation in several animal models. PPAR-γ ligands may also protect the vasculature indirectly by normalizing metabolic abnormalities of the diabetic milieu that increase cardiovascular risk. Activation of PPAR-γ, newly defined in vascular cells, may be a useful approach to protect the vasculature in diabetes.

AN 2001:136312 HCAPLUS <<LOGINID::20080311>>
DN 134:235155
TI Control of vascular cell proliferation and migration by PPAR-γ: A new approach to the macrovascular complications of diabetes
AU Hsueh, Willa A.; Jackson, Simon; Law, Ronald E.
CS Department of Medicine, the Endocrinology, Diabetes, and Hypertension Division, University of California School of Medicine, Los Angeles, CA, 90095-7073, USA
SO Diabetes Care (2001), 24(2), 392-397
CODEN: DICAD2; ISSN: 0149-5992
PB American Diabetes Association, Inc.
DT Journal; General Review
LA English
RE.CNT 82 THERE ARE 82 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L18 ANSWER 5 OF 6 HCAPLUS COPYRIGHT 2008 ACS on STN
TI Peroxisome proliferator-activated receptor γ activators downregulate angiotensin II type 1 receptor in vascular smooth muscle cells
AB Peroxisome proliferator-activated receptor γ (PPARγ) activators, such as troglitazone (Tro), not only improve insulin resistance but also suppress the neointimal formation after balloon injury. However, the precise mechanisms have not been determined Angiotensin II (Ang II) plays crucial roles in the pathogenesis of atherosclerosis, hypertension, and neointimal formation after angioplasty. The authors examined the effect of PPARγ activators on the expression of Ang II type 1 receptor (AT1-R) in cultured vascular smooth muscle cells (VSMCs). AT1-R mRNA and AT1-R protein levels were determined by Northern blot anal. and radioligand binding assay, resp. Natural PPARγ ligand 15-deoxy-Δ12.14-prostaglandin J2, as well as Tro, reduced the AT1-R mRNA expression and the AT1-R protein level. The PPARγ activators also reduced the calcium response of VSMCs to Ang II. PPARγ activators suppressed the AT1-R promoter activity measured by luciferase assay but did not affect the AT1-R mRNA stability, suggesting that the suppression occurs at the transcriptional level. PPARγ activators reduced the AT1-R expression and calcium response to Ang II in VSMCs. Downregulation of AT1-R may contribute to the inhibition of neointimal formation by PPAR γ activators.
AN 2000:759543 HCAPLUS <<LOGINID::20080311>>
DN 134:66617
TI Peroxisome proliferator-activated receptor γ activators downregulate angiotensin II type 1 receptor in vascular smooth muscle cells
AU Takeda, Kotaro; Ichiki, Toshihiro; Tokunou, Tomotake; Funakoshi, Yuko; Iino, Naoko; Hirano, Katsuya; Kanaide, Hideo; Takeshita, Akira
CS Departments of Cardiovascular Medicine, Kyushu University Graduate School of Medical Sciences, Fukuoka, 812-8582, Japan
SO Circulation (2000), 102(15), 1834-1839
CODEN: CIRCAZ; ISSN: 0009-7322
PB Lippincott Williams & Wilkins
DT Journal
LA English
RE.CNT 26 THERE ARE 26 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L18 ANSWER 6 OF 6 HCAPLUS COPYRIGHT 2008 ACS on STN

TI Expression and function of PPARγ in rat and human vascular smooth muscle cells

AB Peroxisome proliferator-activated receptor-γ (PPARγ) is activated by fatty acids, eicosanoids, and insulin-sensitizing thiazolidinediones (TZDs). The TZD troglitazone (TRO) inhibits vascular smooth muscle cell (VSMC) proliferation and migration in vitro and in post-injury intimal hyperplasia. Rat and human VSMCs expressed mRNA and nuclear PPARγ1. Three PPARγ ligands, the TZDs TRO and rosiglitazone and the prostanoid 15-deoxy-Δ12,14-prostaglandin J2 (15d-PGJ2), all inhibited VSMC proliferation and migration. PPARγ was upregulated in rat neointima at 7 days and 14 days after balloon injury and was also present in early human atheroma and precursor lesions. Thus, pharmacol. activation of PPAR.gamma. expressed in VSMCs inhibits their proliferation and migration, potentially limiting restenosis and atherosclerosis. These receptors are upregulated during vascular injury.

AN 2000:240919 HCAPLUS <<LOGINID::20080311>>

DN 133:148479

TI Expression and function of PPARγ in rat and human vascular smooth muscle cells

AU Law, Ronald E.; Goetze, Stephan; Xi, Xiao-Ping; Jackson, Simon; Kawano, Yasuko; Demer, Linda; Fishbein, Michael C.; Meehan, Woerner P.; Hsueh, Willa A.

CS Department of Medicine, University of California at Los Angeles School of Medicine, Los Angeles, CA, 90095, USA

SO Circulation (2000), 101(11), 1311-1318
CODEN: CIRCAZ; ISSN: 0009-7322

PB Lippincott Williams & Wilkins

DT Journal

LA English

RE.CNT 33 THERE ARE 33 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> d his

(FILE 'HOME' ENTERED AT 09:06:15 ON 11 MAR 2008)

FILE 'HCAPLUS' ENTERED AT 09:08:26 ON 11 MAR 2008
L1 13099 S NEOTINTIMA OR RESTENOSIS OR STENT
L2 12209 S (PPAR OR (PEROXISOME PROLIFERATOR-ACTIVATED RECEPTOR))
L3 632628 S PHOSPHATE OR MONOACYLGLYCEROL OR DIACYLGLYCEROL OR PYROPHOSPH
L4 100 S L1 AND L2
L5 1 S L1 AND L2 AND L3
L6 56 S L4 AND (PY<2004 OR AY<2004 OR PRY<2004)
L7 0 S L5 AND (PY<2004 OR AY<2004 OR PRY<2004)

FILE 'STNGUIDE' ENTERED AT 09:08:37 ON 11 MAR 2008

FILE 'HCAPLUS' ENTERED AT 09:08:51 ON 11 MAR 2008

FILE 'STNGUIDE' ENTERED AT 09:08:51 ON 11 MAR 2008

FILE 'HCAPLUS' ENTERED AT 09:10:57 ON 11 MAR 2008
L8 14301 S NEOINTIMA OR RESTENOSIS OR STENT
L9 1527 S (PPAR OR (PEROXISOME PROLIFERATOR-ACTIVATED RECEPTOR))(4A)(IN
L10 29 S L8 AND L9
L11 19 S L10 AND (PY<2004 OR AY<2004 OR PRY<2004)

FILE 'STNGUIDE' ENTERED AT 09:11:04 ON 11 MAR 2008

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     FILE 'HCAPLUS' ENTERED AT 09:11:13 ON 11 MAR 2008

     FILE 'STNGUIDE' ENTERED AT 09:11:15 ON 11 MAR 2008

     FILE 'HCAPLUS' ENTERED AT 09:42:17 ON 11 MAR 2008
L12          8522 S NEOINTIMA OR NEOINTIMAL OR STENT
L13            45 S L2 AND L12
L14            16 S L9 AND L12
L15        550214 S L3 AND (PY<2004 OR AY<2004 OR PRY<2004)
L16        550214 S L3 AND (PY<2004 OR AY<2004 OR PRY<2004)

     FILE 'STNGUIDE' ENTERED AT 09:42:28 ON 11 MAR 2008

     FILE 'HCAPLUS' ENTERED AT 09:42:49 ON 11 MAR 2008
L17           15 S L13 AND (PY<2004 OR AY<2004 OR PRY<2004)
L18            6 S L14 AND (PY<2004 OR AY<2004 OR PRY<2004)

     FILE 'STNGUIDE' ENTERED AT 09:42:55 ON 11 MAR 2008

     FILE 'HCAPLUS' ENTERED AT 09:43:04 ON 11 MAR 2008

     FILE 'STNGUIDE' ENTERED AT 09:43:04 ON 11 MAR 2008

=> log hold
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                         ENTRY    SESSION
FULL ESTIMATED COST                                       0.06      95.99

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)          SINCE FILE      TOTAL
                                                         ENTRY    SESSION
CA SUBSCRIBER PRICE                                       0.00     -20.80

 SESSION WILL BE HELD FOR 120 MINUTES
STN INTERNATIONAL SESSION SUSPENDED AT 09:43:10 ON 11 MAR 2008

Connecting via Winsock to STN



Welcome to STN International!  Enter x:x

LOGINID:SSPTAEXO1623

PASSWORD:
 * * * * * * RECONNECTED TO STN INTERNATIONAL * * * * * *
SESSION RESUMED IN FILE 'STNGUIDE' AT 09:58:34 ON 11 MAR 2008
FILE 'STNGUIDE' ENTERED AT 09:58:34 ON 11 MAR 2008
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COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                         ENTRY    SESSION
FULL ESTIMATED COST                                       0.06      95.99

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)          SINCE FILE      TOTAL
                                                         ENTRY    SESSION
CA SUBSCRIBER PRICE                                       0.00     -20.80

=> file registry
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                         ENTRY    SESSION
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FULL ESTIMATED COST                                    0.06      95.99

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)       SINCE FILE      TOTAL
                                                    ENTRY     SESSION
CA SUBSCRIBER PRICE                                  0.00      -20.80

FILE 'REGISTRY' ENTERED AT 09:58:41 ON 11 MAR 2008
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Property values tagged with IC are from the ZIC/VINITI data file
provided by InfoChem.

STRUCTURE FILE UPDATES:   10 MAR 2008  HIGHEST RN 1007341-18-5
DICTIONARY FILE UPDATES:  10 MAR 2008  HIGHEST RN 1007341-18-5

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TSCA INFORMATION NOW CURRENT THROUGH January 9, 2008.

  Please note that search-term pricing does apply when
  conducting SmartSELECT searches.

REGISTRY includes numerically searchable data for experimental and
predicted properties as well as tags indicating availability of
experimental property data in the original document.  For information
on property searching in REGISTRY, refer to:

http://www.cas.org/support/stngen/stndoc/properties.html

=> exp lysophosphatid/cn
E1            1     LYSOPEPTIN B/CN
E2            1     LYSOPHOPHOLIPASE (SACCHAROMYCES CEREVISIAE STRAIN S288C GENE
                     PLB1)/CN
E3            0 --> LYSOPHOSPHATID/CN
E4            1     LYSOPHOSPHATIDALCHOLINES, Γ-O-1-HEXADECENYL-A/CN
E5            1     LYSOPHOSPHATIDALCHOLINES, Γ-O-1-PENTADECENYL-A/C
                    N
E6            1     LYSOPHOSPHATIDALETHANOLAMINE ACYLTRANSFERASE/CN
E7            1     LYSOPHOSPHATIDASE/CN
E8            1     LYSOPHOSPHATIDATE ACYLTRANSFERASE/CN
E9            1     LYSOPHOSPHATIDATE LYSOPHOSPHOLIPASE A1/CN
E10           1     LYSOPHOSPHATIDATE PHOSPHATASE/CN
E11           1     LYSOPHOSPHATIDATE PHOSPHOHYDROLASE/CN
E12           1     LYSOPHOSPHATIDATE RECEPTOR (HUMAN JURKAT T CELL GENE EDG7)/C
                    N

=> exp lysophosphatidic/cn
E1            1     LYSOPHOSPHATIDE ACYLTRANSFERASE/CN
E2            1     LYSOPHOSPHATIDES, LYSOCARDIOLIPINS, CATTLE HEART, SODIUM SAL
                    TS/CN
E3            0 --> LYSOPHOSPHATIDIC/CN
E4            1     LYSOPHOSPHATIDIC ACID ACYL TRANSFERASE (HUMAN SEQUENCE HOMOL
                    OG)/CN
E5            1     LYSOPHOSPHATIDIC ACID ACYLTRANSFERASE/CN
E6            1     LYSOPHOSPHATIDIC ACID ACYLTRANSFERASE (BDELLOVIBRIO BACTERIO
                    VORUS STRAIN HD100)/CN
E7            1     LYSOPHOSPHATIDIC ACID ACYLTRANSFERASE (HUMAN CLONE MGC:59880
                     IMAGE:6649895)/CN
E8            1     LYSOPHOSPHATIDIC ACID ACYLTRANSFERASE (HUMAN CLONE MGC:71254
```

```
                    IMAGE:6577569)/CN
E9            1     LYSOPHOSPHATIDIC ACID ACYLTRANSFERASE (HUMAN ISOENZYME LPAAT
                    -Z)/CN
E10           1     LYSOPHOSPHATIDIC ACID ACYLTRANSFERASE (KUENENIA STUTTGARTIEN
                    SIS GENE NLAB)/CN
E11           1     LYSOPHOSPHATIDIC ACID ACYLTRANSFERASE (MOUSE STRAIN FVB/N CL
                    ONE MGC:28958 IMAGE:4457846)/CN
E12           1     LYSOPHOSPHATIDIC ACID ACYLTRANSFERASE (ORYZA SATIVA JAPONICA
                     GENE OSJNBA0017E08.6)/CN

=> exp lysophosphatidic acid/cn
E1            1     LYSOPHOSPHATIDE ACYLTRANSFERASE/CN
E2            1     LYSOPHOSPHATIDES, LYSOCARDIOLIPINS, CATTLE HEART, SODIUM SAL
                    TS/CN
E3            0 --> LYSOPHOSPHATIDIC ACID/CN
E4            1     LYSOPHOSPHATIDIC ACID ACYL TRANSFERASE (HUMAN SEQUENCE HOMOL
                    OG)/CN
E5            1     LYSOPHOSPHATIDIC ACID ACYLTRANSFERASE/CN
E6            1     LYSOPHOSPHATIDIC ACID ACYLTRANSFERASE (BDELLOVIBRIO BACTERIO
                    VORUS STRAIN HD100)/CN
E7            1     LYSOPHOSPHATIDIC ACID ACYLTRANSFERASE (HUMAN CLONE MGC:59880
                     IMAGE:6649895)/CN
E8            1     LYSOPHOSPHATIDIC ACID ACYLTRANSFERASE (HUMAN CLONE MGC:71254
                     IMAGE:6577569)/CN
E9            1     LYSOPHOSPHATIDIC ACID ACYLTRANSFERASE (HUMAN ISOENZYME LPAAT
                    -Z)/CN
E10           1     LYSOPHOSPHATIDIC ACID ACYLTRANSFERASE (KUENENIA STUTTGARTIEN
                    SIS GENE NLAB)/CN
E11           1     LYSOPHOSPHATIDIC ACID ACYLTRANSFERASE (MOUSE STRAIN FVB/N CL
                    ONE MGC:28958 IMAGE:4457846)/CN
E12           1     LYSOPHOSPHATIDIC ACID ACYLTRANSFERASE (ORYZA SATIVA JAPONICA
                     GENE OSJNBA0017E08.6)/CN

=> exp lpa/cn
E1            1     LP85 (277-PROLINE) (HUMAN PRECURSOR)/CN
E2            1     LP85 (279-PROLINE) (HUMAN PRECURSOR),/CN
E3            1 --> LPA/CN
E4            1     LPA 170/CN
E5            1     LPA 2/CN
E6            1     LPA 210/CN
E7            1     LPA 2SC/CN
E8            1     LPA 3/CN
E9            1     LPA 3500/CN
E10           1     LPA 39/CN
E11           1     LPA 47/CN
E12           1     LPA-2 RECEPTOR (HUMAN LYSOPHOSPHATIDIC ACID RECEPTOR 2)/CN

=> s e3
L19           1 LPA/CN

=> d l19

L19  ANSWER 1 OF 1  REGISTRY  COPYRIGHT 2008 ACS on STN
RN   142-54-1  REGISTRY
ED   Entered STN:  16 Nov 1984
CN   Dodecanamide, N-(2-hydroxypropyl)-  (CA INDEX NAME)
OTHER NAMES:
CN   2-Hydroxypropyllauramide
CN   Alkamide LIPA
CN   Amisol PLME
CN   Clindrol 101LI
```

CN Clindrol 102LI
CN Comperlan LP
CN Cyclomide LP
CN Lauramide MIPA
CN Lauric acid isopropanolamide
CN Lauric acid monoisopropanolamide
CN Lauric isopropanolamide
CN Lauric monoisopropanolamide
CN Lauroyl isopropanolamide
CN Lauryl isopropanolamide
CN Lauryl monoisopropanolamide
CN LIPA
CN LPA
CN N-(β-Hydroxypropyl)lauramide
CN N-(2-Hydroxy-1-propyl)lauramide
CN N-(2-Hydroxypropyl)dodecanamide
CN Profan AD31
CN Stafoam LIPA
CN Steinamid IPL 203
CN Ultrapole L
MF C15 H31 N O2
CI COM
LC STN Files: BEILSTEIN*, BIOSIS, CA, CAOLD, CAPLUS, CHEMLIST, CSCHEM, IFICDB, IFIPAT, IFIUDB, MSDS-OHS, TOXCENTER, USPAT2, USPATFULL, USPATOLD
(*File contains numerically searchable property data)
Other Sources: DSL**, EINECS**, TSCA**
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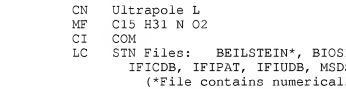

PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

143 REFERENCES IN FILE CA (1907 TO DATE)
2 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
143 REFERENCES IN FILE CAPLUS (1907 TO DATE)
15 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

=> file stnguide

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	8.07	104.06
DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-20.80

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FILE CONTAINS CURRENT INFORMATION.
LAST RELOADED: Mar 7, 2008 (20080307/UP).

=> file hcaplus

```
COST IN U.S. DOLLARS                                    SINCE FILE      TOTAL
                                                          ENTRY    SESSION
FULL ESTIMATED COST                                         0.24     104.30

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)              SINCE FILE      TOTAL
                                                          ENTRY    SESSION
CA SUBSCRIBER PRICE                                         0.00     -20.80

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```



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FILE COVERS 1907 - 11 Mar 2008  VOL 148 ISS 11
FILE LAST UPDATED: 10 Mar 2008  (20080310/ED)

New CAS Information Use Policies, enter HELP USAGETERMS for details.

 This file contains CAS Registry Numbers for easy and accurate
 substance identification.

=> s lysophosphatidic acid

          3386 LYSOPHOSPHATIDIC
       4543306 ACID
L20       2675 LYSOPHOSPHATIDIC ACID
                 (LYSOPHOSPHATIDIC(W)ACID)

=> s l20 and l2

L21         38 L20 AND L2

=> s l10 and l9

L22         29 L10 AND L9

=> s l21 and l12

L23          4 L21 AND L12

=> s l22 and l12

L24         12 L22 AND L12

=> s l23 and (PY<2004 or AY<2004 or PRY<2004)

      23979567 PY<2004
       4765121 AY<2004
       4243738 PRY<2004
L25          1 L23 AND (PY<2004 OR AY<2004 OR PRY<2004)
```

```
=> s 124 and (PY<2004 or AY<2004 or PRY<2004)

      23979567 PY<2004
       4765121 AY<2004
       4243738 PRY<2004
L26           4 L24 AND (PY<2004 OR AY<2004 OR PRY<2004)

=> file stnguide

COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                         ENTRY    SESSION
FULL ESTIMATED COST                                       2.69     106.99

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)          SINCE FILE      TOTAL
                                                         ENTRY    SESSION
CA SUBSCRIBER PRICE                                       0.00     -20.80

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FILE CONTAINS CURRENT INFORMATION.
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=> file hcaplus
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                         ENTRY    SESSION
FULL ESTIMATED COST                                       0.06     107.05

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)          SINCE FILE      TOTAL
                                                         ENTRY    SESSION
CA SUBSCRIBER PRICE                                       0.00     -20.80

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FILE COVERS 1907 - 11 Mar 2008  VOL 148 ISS 11
FILE LAST UPDATED: 10 Mar 2008  (20080310/ED)

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 This file contains CAS Registry Numbers for easy and accurate
 substance identification.

=> s 120 and 19

L27           9 L20 AND L9
```

```
=> s l27 and l12

L28           2 L27 AND L12

=> s l28 and (PY<2004 or AY<2004 or PRY<2004)

      23979567 PY<2004
       4765121 AY<2004
       4243738 PRY<2004
L29           1 L28 AND (PY<2004 OR AY<2004 OR PRY<2004)

=> file stnguide

COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                       ENTRY    SESSION
FULL ESTIMATED COST                                     2.69     109.74

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)          SINCE FILE      TOTAL
                                                       ENTRY    SESSION
CA SUBSCRIBER PRICE                                     0.00     -20.80

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=> file hcaplus
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                       ENTRY    SESSION
FULL ESTIMATED COST                                     0.06     109.80

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)          SINCE FILE      TOTAL
                                                       ENTRY    SESSION
CA SUBSCRIBER PRICE                                     0.00     -20.80

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FILE LAST UPDATED: 10 Mar 2008  (20080310/ED)

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 This file contains CAS Registry Numbers for easy and accurate
 substance identification.
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=> s l21 and (PY<2004 or AY<2004 or PRY<2004)

      23979567 PY<2004
       4765121 AY<2004
       4243738 PRY<2004
L30           7 L21 AND (PY<2004 OR AY<2004 OR PRY<2004)

=> s l27 and (PY<2004 or AY<2004 or PRY<2004)

      23979567 PY<2004
       4765121 AY<2004
       4243738 PRY<2004
L31           1 L27 AND (PY<2004 OR AY<2004 OR PRY<2004)

=> file stnguide

COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                       ENTRY    SESSION
FULL ESTIMATED COST                                     2.69     112.49

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)          SINCE FILE      TOTAL
                                                       ENTRY    SESSION
CA SUBSCRIBER PRICE                                     0.00     -20.80

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FILE CONTAINS CURRENT INFORMATION.
LAST RELOADED: Mar  7, 2008 (20080307/UP).

=> d l30 1-7 ti abs bib
YOU HAVE REQUESTED DATA FROM FILE 'HCAPLUS' - CONTINUE? (Y)/N:y



L30  ANSWER 1 OF 7  HCAPLUS  COPYRIGHT 2008 ACS on STN
TI   Gene expression profiles and biomarkers for the detection of
     depression-related and other disease-related gene transcripts in blood
AB   The present invention is directed to detection and measurement of gene
     transcripts and their equivalent nucleic acid products in blood.  Specifically
     provided is anal. performed on a drop of blood for detecting, diagnosing,
     and monitoring diseases, and in particular mental depression, using
     gene-specific and/or tissue-specific primers.  Affymetrix Human Genome
     U133 and ChondroChip microarrays were used to detect differentially
     expressed gene transcripts in hypertension, obesity, allergy, systemic
     steroids, coronary artery disease, diabetes type 2, hyperlipidemia, lung
     disease, bladder cancer, rheumatoid arthritis, osteoarthritis, liver
     cancer, schizophrenia, Chagas disease, asthma, and manic depression
     syndrome.  The present invention describes methods by which delineation of
     the sequence and/or quantitation of the expression levels of
     disease-specific genes allows for an immediate and accurate
     diagnostic/prognostic test for disease or to assess the effect of a
     particular treatment regimen.
AN   2005:1997  HCAPLUS <<LOGINID::20080311>>
DN   142:111841
TI   Gene expression profiles and biomarkers for the detection of
     depression-related and other disease-related gene transcripts in blood
IN   Liew, Choong-Chin
```

PA Chondrogene Limited, Can.
SO U.S. Pat. Appl. Publ., 154 pp., Cont.-in-part of U.S. Ser. No. 802,875.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 33

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 2004265868	A1	20041230	US 2004-812702	20040330 <--
	US 2004014059	A1	20040122	US 2002-268730	20021009 <--
	US 2007031841	A1	20070208	US 2003-601518	20030620 <--
	US 2006134635	A1	20060622	US 2004-802875	20040312 <--
	US 2005191637	A1	20050901	US 2004-803737	20040318 <--
	US 2005196762	A1	20050908	US 2004-803759	20040318 <--
	US 2005196763	A1	20050908	US 2004-803857	20040318 <--
	US 2005196764	A1	20050908	US 2004-803858	20040318 <--
	US 2005208505	A1	20050922	US 2004-803648	20040318 <--
PRAI	US 1999-115125P	P	19990106	<--	
	US 2000-477148	B1	20000104	<--	
	US 2002-268730	A2	20021009	<--	
	US 2003-601518	A2	20030620	<--	
	US 2004-802875	A2	20040312		
	US 2001-271955P	P	20010228	<--	
	US 2001-275017P	P	20010312	<--	
	US 2001-305340P	P	20010713	<--	
	US 2002-85783	A2	20020228	<--	

L30 ANSWER 2 OF 7 HCAPLUS COPYRIGHT 2008 ACS on STN
TI Lysophosphatidic acid analogs and inhibition of neointima formation
AB The phospholipid growth factor lysophosphatidic acids (LPAs) containing unsatd. fatty acids (18:1, 18:2 and 20:4) and fatty alcs. containing hydrocarbon chains with more than 4 carbons were capable of inducing a rapid formation of neointima, an initial step in the development of atherosclerotic plaque. LPAs with saturated fatty acids did not induce neointima formation. A Peroxisome Proliferator-Activated Receptors gamma (PPAR.gamma.)-specific agonist Rosiglitasone also induced a profound formation of neointima. GW9662, a selective and irreversible antagonist of PPAR.gamma., abolished LPA- and Rosiglitazone-induced neointima formation, indicating that LPA-induced neointima formation requires the activation of PPAR.gamma.. These data suggest that LPA analogs that bind to but do not activate downstream signaling of PPAR.gamma. or antagonists of PPAR.gamma. that inhibit PPAR.gamma. signaling would be useful in the prevention and/or treatment of neointima formation and atherosclerosis.
AN 2004:857161 HCAPLUS <<LOGINID::20080311>>
DN 141:343506
TI Lysophosphatidic acid analogs and inhibition of neointima formation
IN Tigyi, Gabor; Baker, Daniel L.; Zhang, Chunxiang
PA USA
SO U.S. Pat. Appl. Publ., 23 pp.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 2004204383	A1	20041014	US 2004-821739	20040409 <--
	AU 2004229467	A1	20041028	AU 2004-229467	20040409 <--

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     AU 2004229467         B2    20070125
     CA 2521189            A1    20041028    CA 2004-2521189        20040409 <--
     WO 2004091496         A2    20041028    WO 2004-US11016        20040409 <--
     WO 2004091496         A3    20050324
         W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH,
             CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD,
             GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC,
             LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI,
             NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY,
             TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
         RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ,
             BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE,
             ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI,
             SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN,
             TD, TG
     EP 1613298            A2    20060111    EP 2004-759365         20040409 <--
         R:  AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
             IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK, HR
     JP 2007525449         T     20070906    JP 2006-509874         20040409 <--
PRAI US 2003-462274P       P     20030411  <--
     WO 2004-US11016       W     20040409


L30  ANSWER 3 OF 7  HCAPLUS  COPYRIGHT 2008 ACS on STN
TI   Lysophosphatidic acid signaling: how a small lipid
     does big things. [Erratum to document cited in CA139:211216]
AB   A review.  The corrected version of Figure 2 is given.
AN   2003:728621  HCAPLUS <<LOGINID::20080311>>
DN   141:68526
TI   Lysophosphatidic acid signaling: how a small lipid
     does big things. [Erratum to document cited in CA139:211216]
AU   Luquain, CelineAnon.; Sciorra, Vicki A.; Morris, Andrew J.
CS   Lineberger Comprehensive Cancer Center, Department of Cell Developmental
     Biology, The University of North Carolina at Chapel Hill, Chapel Hill, NC,
     27699-7090, USA
SO   Trends in Biochemical Sciences (2003), 28(9), 478
     CODEN: TBSCDB; ISSN: 0968-0004
PB   Elsevier Science Ltd.
DT   Journal; General Review
LA   English

L30  ANSWER 4 OF 7  HCAPLUS  COPYRIGHT 2008 ACS on STN
TI   Lysophosphatidic acid signaling: how a small lipid
     does big things
AB   A review.  Lysophosphatidic acid (LPA) promotes
     growth, differentiation, survival and motility in many different cell
     types.  LPA has therefore been suggested to play a central role in a broad
     range of physiol. and pathophysiol. processes, including vascular and
     neuronal function and cancer.  Three closely related G-protein-coupled
     cell-surface receptors mediate some of these effects, but assigning
     specific functions to particular receptor subtypes has been challenging
     and several lines of evidence indicate that other LPA signaling mechanisms
     might exist.  Although the signaling actions of LPA have been studied
     widely, much less is known about how LPA is generated and released into
     the extracellular space, and how its signaling actions are terminated.
     Newly identified enzymes that generate and inactivate LPA have novel roles
     in cancer progression and early development, and a recent study indicates
     that LPA might regulate nuclear gene transcription directly.  These
     findings provide novel insights into mechanisms involved in the synthesis,
     actions and inactivation of LPA, and the proteins involved provide new
     targets that can be exploited to manipulate LPA signaling at both cellular
     and organismal levels.
```

AN 2003:564080 HCAPLUS <<LOGINID::20080311>>
DN 139:211216
TI Lysophosphatidic acid signaling: how a small lipid
does big things
AU Luquain, Celine; Sciorra, Vicki A.; Morris, Andrew J.
CS Lineberger Comprehensive Cancer Center, Department of Cell and
Developmental Biology, The University of North Carolina at Chapel Hill,
Chapel Hill, NC, 27699-7090, USA
SO Trends in Biochemical Sciences (2003), 28(7), 377-383
CODEN: TBSCDB; ISSN: 0968-0004
PB Elsevier Science Ltd.
DT Journal; General Review
LA English
RE.CNT 49 THERE ARE 49 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L30 ANSWER 5 OF 7 HCAPLUS COPYRIGHT 2008 ACS on STN
TI Synthesis of Monofluorinated Analogues of Lysophosphatidic
Acid
AB Lysophosphatidic acid (LPA, 1- or 2-acyl-sn-glycerol
3-phosphate) displays an intriguing cell biol. that is mediated via
interactions both with G-protein coupled seven transmembrane receptors and
with the nuclear hormone receptor PPAR.gamma.. Synthesis and
biol. activities of fluorinated analogs of LPA are still relatively
unknown. In an effort to identify receptor-selective LPA analogs and to
document in detail the structure-activity relationships of fluorinated LPA
isosteres, we describe a series of monofluorinated LPA analogs in which
either the sn-1 or the sn-2 hydroxy group was replaced by fluorine, or the
bridging oxygen in the monophosphate was replaced by an
α-monofluoromethylene (-CHF-) moiety. The sn-1 or sn-2
monofluorinated LPA analogs were enantiospecifically prepared from chiral
protected glycerol synthons, and the α-monofluoromethylene-
substituted LPA analogs were prepared from a racemic epoxide with use of a
hydrolytic kinetic resolution The sn-2 and sn-1 fluoro LPA analogs were
unable to undergo acyl migration, effectively "freezing" them in the
sn-1-O-acyl or sn-2-O-acyl forms, resp. The α-monofluoromethylene
LPA analogs were unique new nonhydrolyzable ligands with surprising
enantiospecific and receptor-specific biol. readouts, with one compound
showing a 1000-fold higher activity than native LPA for one receptor
subtype.
AN 2003:418219 HCAPLUS <<LOGINID::20080311>>
DN 139:133754
TI Synthesis of Monofluorinated Analogues of Lysophosphatidic
Acid
AU Xu, Yong; Qian, Lian; Prestwich, Glenn D.
CS Department of Medicinal Chemistry and The Center for Cell Signaling,
University of Utah, Salt Lake City, UT, 84108-1257, USA
SO Journal of Organic Chemistry (2003), 68(13), 5320-5330
CODEN: JOCEAH; ISSN: 0022-3263
PB American Chemical Society
DT Journal
LA English
OS CASREACT 139:133754
RE.CNT 54 THERE ARE 54 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L30 ANSWER 6 OF 7 HCAPLUS COPYRIGHT 2008 ACS on STN
TI Identification of an intracellular receptor for lysophosphatidic
acid (LPA): LPA is a transcellular PPAR.gamma. agonist.
[Erratum to document cited in CA139:98489]
AB Figure 4 should have appeared in color; the correct figure and its legend

are given.
AN 2003:155856 HCAPLUS <<LOGINID::20080311>>
DN 140:39353
TI Identification of an intracellular receptor for lysophosphatidic acid (LPA): LPA is a transcellular PPAR.gamma. agonist. [Erratum to document cited in CA139:98489]
AU McIntyre, Thomas M.; Pontsler, Aaron V.; Silva, Adriana R.; St. Hilaire, Andy; Xu, Yong; Hinshaw, Jerald C.; Zimmerman, Guy A.; Hama, Kotaro; Aoki, Junken; Arai, Hiroyuki; Prestwich, Glenn D.
CS Program in Human Molecular Biology and Genetics, and Department of Medicine, University of Utah, Salt Lake City, UT, 84112-5330, USA
SO Proceedings of the National Academy of Sciences of the United States of America (2003), 100(4), 2163
CODEN: PNASA6; ISSN: 0027-8424
PB National Academy of Sciences
DT Journal
LA English

L30 ANSWER 7 OF 7 HCAPLUS COPYRIGHT 2008 ACS on STN
TI Identification of an intracellular receptor for lysophosphatidic acid (LPA): LPA is a transcellular PPAR.gamma. agonist
AB Lysophosphatidic acid (LPA) is a pluripotent lipid mediator acting through plasma membrane-associated LPAx receptors that transduce many, but not all, of its effects. We identify peroxisome proliferator-activated receptor γ (PPAR.gamma.) as an intracellular receptor for LPA. The transcription factor PPAR.gamma. is activated by several lipid ligands, but agonists derived from physiol. signaling pathways are unknown. We show that LPA, but not its precursor phosphatidic acid, displaces the drug rosiglitazone from the ligand-binding pocket of PPAR.gamma.. LPA and novel LPA analogs we made stimulated expression of a PPAR-responsive element reporter and the endogenous PPAR.gamma.-controlled gene CD36, and induced monocyte lipid accumulation from oxidized low-d. lipoprotein via the CD36 scavenger receptor. The synthetic LPA analogs were effective PPAR.gamma. agonists, but were poor ones for LPA1, LPA2, or LPA3 receptor transfected cells. Transfection studies in yeast, which lack nuclear hormone and LPAx receptors, show that LPA directly activates PPAR.gamma.. A major growth factor of serum is LPA generated by thrombin-activated platelets, and media from activated platelets stimulated PPAR.gamma. function in transfected RAW264.7 macrophages. This function was suppressed by ectopic LPA-acyltransferase expression. LPA is a physiol. PPAR.gamma. ligand, placing PPAR.gamma. in a signaling pathway, and PPAR.gamma. is the first intracellular receptor identified for LPA. Moreover, LPA produced by stimulated plasma platelets activates PPAR.gamma. in nucleated cells.
AN 2003:43726 HCAPLUS <<LOGINID::20080311>>
DN 139:98489
TI Identification of an intracellular receptor for lysophosphatidic acid (LPA): LPA is a transcellular PPAR.gamma. agonist
AU McIntyre, Thomas M.; Pontsler, Aaron V.; Silva, Adriana R.; St. Hilaire, Andy; Xu, Yong; Hinshaw, Jerald C.; Zimmerman, Guy A.; Hama, Kotaro; Aoki, Junken; Arai, Hiroyuki; Prestwich, Glenn D.
CS Program in Human Molecular Biology and Genetics, and Department of Medicine, University of Utah, Salt Lake City, UT, 84112-5330, USA
SO Proceedings of the National Academy of Sciences of the United States of America (2003), 100(1), 131-136
CODEN: PNASA6; ISSN: 0027-8424
PB National Academy of Sciences
DT Journal

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LA   English
RE.CNT  45    THERE ARE 45 CITED REFERENCES AVAILABLE FOR THIS RECORD
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          1938 NEOINTIMA
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          5656 STENT
          3386 LYSOPHOSPHATIDIC
       4543306 ACID
          2675 LYSOPHOSPHATIDIC ACID
                 (LYSOPHOSPHATIDIC(W)ACID)
L32          9 L12 AND L20
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=> d l32 1-9 ti abs bib

L32 ANSWER 1 OF 9 CAPLUS COPYRIGHT 2008 ACS on STN
TI Enhanced sterol response element-binding protein in postintervention restenotic blood vessels plays an important role in vascular smooth muscle proliferation
AB Postintervention restenosis (PIRS) after balloon angioplasty or stent implantation is a limitation for these interventional procedures even with the advent of new drug-eluting stents. Sterol regulatory element-binding proteins (SREBP) are transcription factors governing cellular lipid biosynthesis and thus critical in the regulation of the lipid-rich cell membranes. PIRS following injury results partially from newly proliferating cells expressing vascular smooth muscle cell (VSMC) markers. Platelet-derived growth factor (PDGF), lysophosphatidic acid (LPA) and α1-adrenergic receptor stimulation are well recognized diverse mitogens for VSMC activation in PIRS. We examined whether PDGF, LPA and α1-adrenergic receptor stimulation with phenylephrine (PE) regulate SREBP expression and subsequently, VSMC proliferation. Our results show that PDGF, LPA and PE upregulate SREBP-1 in a time- and dose-dependent manner. PDGF, LPA and PE-mediated proliferation is dependent on SREBP since inhibition of SREBP expression using targeted knockdown of the SREBP precursor SREBP activating protein (SCAP) by siRNA led to an attenuation of SREBP expression and decreased PDGF, LPA and PE induced proliferation. In two different in vivo PIRS models we found that SREBP-1 was enhanced in the injured blood vessel wall, especially within the neointima and co-localized with α-smooth muscle actin pos. cells. Thus, SREBP is enhanced in the vessel wall following PIRS and is important in the regulation of pro-hyperplasia mol. signaling. SREBP inhibition may be a powerful tool to limit PIRS.
AN 2008:26354 CAPLUS <<LOGINID::20080311>>
TI Enhanced sterol response element-binding protein in postintervention restenotic blood vessels plays an important role in vascular smooth muscle proliferation
AU Zhou, Rui-Hai; Pesant, Stephanie; Cohn, Heather I.; Eckhart, Andrea D.
CS Eugene Feiner Laboratory of Vascular Biology and Thrombosis, Center for Translational Medicine, Thomas Jefferson University, Philadelphia, PA, 19107
SO Life Sciences (2008), 82(3-4), 174-181
CODEN: LIFSAK; ISSN: 0024-3205
PB Elsevier B.V.
DT Journal
LA English

L32 ANSWER 2 OF 9 CAPLUS COPYRIGHT 2008 ACS on STN
TI Vascular smooth muscle migration and proliferation in response to lysophosphatidic acid (LPA) is mediated by LPA receptors coupling to Gq
AB Many G protein-coupled receptors can couple to multiple G proteins to convey their intracellular signaling cascades. The receptors for lysophosphatidic acid (LPA) possess this ability. LPA receptors are important mediators of a wide variety of biol. actions including cell migration, proliferation and survival which are processes that can all have a considerable impact on vascular smooth muscle (VSM) and blood vessels. To date, confirmation of G proteins involved has mostly relied on the inhibition of Gi-mediated signaling via pertussis toxin (PTx). We were interested in the specific involvement of LPA-Gq-mediated signaling therefore we isolated aorta VSM cells (VSMCs) from transgenic mice that express a peptide inhibitor of Gq, GqI, exclusively in VSM. We detected both LPA1 and LPA2 receptor expression in mouse VSM whereas LPA1 and LPA3 were expressed in rat VSM. SM22-GqI did

not alter LPA-induced migration but it was sufficient to attenuate LPA-induced proliferation. GqI expression also attenuated LPA-induced ERK1/2 and Akt activation by 40-50%. To test the feasibility of this peptide as a potential therapeutic agent, we also generated adenovirus encoding the GqI. Transient expression of GqI was capable of inhibiting both LPA-induced migration and proliferation of VSMCs isolated from rat and mouse. Furthermore, ERK activation in response to LPA was also attenuated in VSMCs with Adv-GqI. Therefore, LPA receptors couple to Gq in VSMC and mediate migration and proliferation which may be mediated through activation of ERK1/2 and Akt. Our data also suggest that both chronic and transient expression of the GqI peptide is an effective strategy to lower Gq-mediated LPA signaling and may be a successful therapeutic strategy to combat diseases with enhanced VSM growth such as occurs following angioplasty or stent implantation.

AN 2006:847306 CAPLUS <<LOGINID::20080311>>
DN 145:502708
TI Vascular smooth muscle migration and proliferation in response to lysophosphatidic acid (LPA) is mediated by LPA receptors coupling to Gq
AU Kim, Jihee; Keys, Janelle R.; Eckhart, Andrea D.
CS Center for Translational Medicine, Thomas Jefferson University, Philadelphia, PA, 19107, USA
SO Cellular Signalling (2006), 18(10), 1695-1701
CODEN: CESIEY; ISSN: 0898-6568
PB Elsevier B.V.
DT Journal
LA English
RE.CNT 25 THERE ARE 25 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L32 ANSWER 3 OF 9 CAPLUS COPYRIGHT 2008 ACS on STN
TI 3-D Database Searching for the Identification of Novel LPA1 Antagonists
AB Lysophosphatidic acid (LPA) is a phospholipid growth factor which is involved in various biol. signaling pathways. Though once thought to have only structural functions, the involvement of LPA in biol. signaling is now clear. LPA influences cell differentiation, survival and motility. LPA can initiate neointima formation, which may lead to cardiovascular disease. LPA is known to be an agonist of four cell-surface G protein coupled receptors (GPCR), LPA 1-4 and one nuclear receptor, the peroxisome proliferator activated receptor gamma (PPARα). A pharmacophore model, representing the min. structural elements necessary to define an antagonist for the LPA1 receptor, has been developed and utilized for searching the National Cancer Institute 3-D database. Approx. 250 compds., which resulted as hits from these searches, have been docked into the LPA1 receptor model. Six compds. which formed promising complexes with the receptor have been tested for antagonist activity. Of these, three showed weak agonism of the LPA1 receptor and two showed antagonism of LPA3 with micromolar potency. Ten addnl. compds. have been requested from NCI for testing purposes. Results from these studies will assist in further refining the LPA1 receptor model and in identifying novel structures as therapeutic leads.
AN 2005:1224719 CAPLUS <<LOGINID::20080311>>
TI 3-D Database Searching for the Identification of Novel LPA1 Antagonists
AU Perygin, Donna H.
CS Chemistry, University of Memphis, Memphis, TN, 38152, USA
SO Abstracts, 57th Southeast/61st Southwest Joint Regional Meeting of the American Chemical Society, Memphis, TN, United States, November 1-4 (2005), NOV04-029 Publisher: American Chemical Society, Washington, D. C.
CODEN: 69HOKM
DT Conference; Meeting Abstract
LA English

L32 ANSWER 4 OF 9 CAPLUS COPYRIGHT 2008 ACS on STN
TI High-throughput Screening for LPA3 Antagonist Selectivity
AB Lysophosphatidic acid(LPA) activates various extracellular and intracellular responses, such as cell proliferation, migration, adhesion, survival, and differentiation. LPA produces theses responses by acting as an agonist for three G-protein coupled receptors(GPCR), LPA. LPA responses are involved in numerous diseases such as prostate cancer, breast cancer, and cardiovascular disease. One area of interest to our group is LPA's role in cardiovascular disease. LPA is one of the culprits responsible for cardiovascular disease. In cardiovascular disease, LPA stimulates platelets and formation of neointima. LPA is involved in plaque rupture and thrombus formation. LPA1 and LPA3 antagonists both inhibit platelet shape change. Identification of selective LPA3 antagonists has the potential to aid the development of new leads for further understanding LPA's role in disease. In our current study we have developed a pharmacophore model based on known LPA3 antagonists that can be used to rapidly screen a database for structurally distinct lead compds. These potential hits can then be studied computationally as well as exptl. Computationally the database hits are rigidly docked; they are then qual. analyzed for potential as new leads. Several non-lipid antagonists with sub-micromolar potency have been identified.
AN 2005:1224688 CAPLUS <<LOGINID::20080311>>
TI High-throughput Screening for LPA3 Antagonist Selectivity
AU Fells, James, Sr.; Parrill, Abby L.
CS Department of Chemistry, University of Memphis, Memphis, TN, 38152-3550, USA
SO Abstracts, 57th Southeast/61st Southwest Joint Regional Meeting of the American Chemical Society, Memphis, TN, United States, November 1-4 (2005), NOV04-004 Publisher: American Chemical Society, Washington, D. C. CODEN: 69HOKM
DT Conference; Meeting Abstract
LA English

L32 ANSWER 5 OF 9 CAPLUS COPYRIGHT 2008 ACS on STN
TI Lysophospholipid receptors
AB A review. The lysophospholipids (LPLs) include lysophosphatidic acid (radyl-lyso-glycerophosphate), 2,3-cyclic phosphatidic acid, 1-alkyl-2-acetyl-glycero-3-phosphate, sphingosine 1-phosphate, dihydro-sphingosine-1-phosphate, sphingosylphosphorylcholine (lysosphingomyelin), and lysophosphatidylcholine. LPLs exert many of their biol. effects through specific plasma membrane and/or intracellular receptors. LPLs are abundantly present in biol. fluids and many of them are generated through stimulus-coupled activation of biochem. pathways. With only very few exceptions (e.g. RH7777 hepatoma, Sf9 insect, and Saccharomyces cerevisiae cells), most cells are responsive to one or more LPLs, indicating a widespread expression of their receptors. LPLs promote cell survival, exert mitogenic/antimitogenic regulation of the cell cycle, affect cell shape and enhance/inhibit cell motility, regulate organotypic differentiation, modulate immunol. responses, and regulate Ca2+ homeostasis. In a pathol. context, LPLs have been shown to play a role in tumor cell invasion, angiogenesis, neointima formation, development of the heart ventricles, chemotherapeutic and radiation resistance, facial dysmorphism, nociception, and suckling behavior. The current understanding of lysophospholipid biol. is very limited and the present understanding of their role in disease is rudimentary.
AN 2005:103923 CAPLUS <<LOGINID::20080311>>
DN 143:21510
TI Lysophospholipid receptors
AU Tigyi, Gabor J.

CS University of Tennessee Health Sciences Center, Memphis, TN, USA
SO Encyclopedia of Biological Chemistry (2004), Volume 2, 602-604.
Editor(s): Lennarz, William J.; Lane, M. Daniel. Publisher: Elsevier Ltd., Oxford, UK.
CODEN: 69GLBX; ISBN: 0-12-443710-9
DT Conference; General Review
LA English
RE.CNT 11 THERE ARE 11 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L32 ANSWER 6 OF 9 CAPLUS COPYRIGHT 2008 ACS on STN
TI Lysophosphatidic acid analogs and inhibition of neointima formation
AB The phospholipid growth factor lysophosphatidic acids (LPAs) containing unsatd. fatty acids (18:1, 18:2 and 20:4) and fatty alcs. containing hydrocarbon chains with more than 4 carbons were capable of inducing a rapid formation of neointima, an initial step in the development of atherosclerotic plaque. LPAs with saturated fatty acids did not induce neointima formation. A Peroxisome Proliferator-Activated Receptors gamma (PPARγ)-specific agonist Rosiglitasone also induced a profound formation of neointima. GW9662, a selective and irreversible antagonist of PPARγ, abolished LPA- and Rosiglitazone-induced neointima formation, indicating that LPA-induced neointima formation requires the activation of PPARγ. These data suggest that LPA analogs that bind to but do not activate downstream signaling of PPARγ or antagonists of PPARγ that inhibit PPARγ signaling would be useful in the prevention and/or treatment of neointima formation and atherosclerosis.
AN 2004:857161 CAPLUS <<LOGINID::20080311>>
DN 141:343506
TI Lysophosphatidic acid analogs and inhibition of neointima formation
IN Tigyi, Gabor; Baker, Daniel L.; Zhang, Chunxiang
PA USA
SO U.S. Pat. Appl. Publ., 23 pp.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 2004204383	A1	20041014	US 2004-821739	20040409
	AU 2004229467	A1	20041028	AU 2004-229467	20040409
	AU 2004229467	B2	20070125		
	CA 2521189	A1	20041028	CA 2004-2521189	20040409
	WO 2004091496	A2	20041028	WO 2004-US11016	20040409
	WO 2004091496	A3	20050324		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	EP 1613298	A2	20060111	EP 2004-759365	20040409
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK, HR				

	JP 2007525449	T	20070906	JP 2006-509874	20040409
PRAI	US 2003-462274P	P	20030411		
	WO 2004-US11016	W	20040409		

L32 ANSWER 7 OF 9 CAPLUS COPYRIGHT 2008 ACS on STN
TI Thrombogenic and atherogenic activities of lysophosphatidic acid
AB A review. Lysophosphatidic acid (LPA) has been identified as a biol. active lipid in mildly-oxidized LDL, human atherosclerotic lesions, and the supernatant of activated platelets. The evidence that LPA has thrombogenic and atherogenic activities has increased substantially in recent years. Supporting the thrombogenic activity of LPA, anal. of the core region of human carotid plaques revealed recently the presence of alkyl- and acyl-mol. species from LPA with high platelet-activating potency (16:0 alkyl-LPA, 20:4 acyl-LPA). LPA, lipid exts. of atherosclerotic plaques, and the lipid-rich core elicited shape change and, in synergy with other platelet stimuli, aggregation of isolated platelets. This effect was completely abrogated by prior incubation of platelets with LPA receptor antagonists. Furthermore, LPA at concns. approaching those found in vivo, induced platelet shape change, aggregation, and platelet-monocyte aggregate formation in blood. LPA-stimulated platelet aggregation was mediated by the ADP-stimulated activation of the P2Y1 and P2Y12 receptors. Supporting its atherogenic activity, LPA is a mitogen and motogen to vascular smooth muscle cells (VSMCs) and an activator of endothelial cells and macrophages. Recently, LPA has been identified as an agonist of the peroxisome proliferator activating receptor γ(PPARγ), which is a key regulator of atherogenesis. LPA elicits progressive neointima formation, which is fully abolished by GW9662, an antagonist of PPARγ. We propose that LPA plays a central role in eliciting vascular remodeling and atherogenesis. Furthermore, upon rupture of lipid-rich atherosclerotic plaques, LPA may trigger platelet aggregation and intra-arterial thrombus formation. Antagonists of LPA receptors might be useful in preventing LPA-elicited thrombus formation and neointima formation in patients with cardiovascular diseases.
AN 2004:654161 CAPLUS <<LOGINID::20080311>>
DN 141:171305
TI Thrombogenic and atherogenic activities of lysophosphatidic acid
AU Siess, Wolfgang; Tigyi, Gabor
CS Institute for Prevention of Cardiovascular Diseases, University of Munich, Germany
SO Journal of Cellular Biochemistry (2004), 92(6), 1086-1094
CODEN: JCEBD5; ISSN: 0730-2312
PB Wiley-Liss, Inc.
DT Journal; General Review
LA English
RE.CNT 46 THERE ARE 46 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L32 ANSWER 8 OF 9 CAPLUS COPYRIGHT 2008 ACS on STN
TI Lysophosphatidic acid induces neointima formation through PPARγ activation
AB Neointimal lesions are characterized by accumulation of cells within the arterial wall and are a prelude to atherosclerotic disease. Here the authors report that a brief exposure to either alkyl ether analogs of the growth factor-like phospholipid lysophosphatidic acid (LPA), products generated during the oxidative modification of low d. lipoprotein, or to unsatd. acyl forms of LPA induce progressive formation of neointima in vivo in a rat carotid artery model.

This effect is completely inhibited by the peroxisome proliferator-activated receptor (PPAR)γ antagonist GW9662 and mimicked by PPARγ agonists Rosiglitazone and 1-O-hexadecyl-2-azeleoylphosphatidylcholine. In contrast, stearoyloxovalerylphosphatidylcholine, a PPARα agonist and the polypeptides epidermal growth factor, platelet-derived growth factor, and vascular endothelial growth factor failed to elicit neointima. The structure-activity relation for neointima induction by LPA analogs in vivo is identical to that of PPARγ activation in vitro and disparate from that of LPA G protein-coupled receptor activation. Neointima-inducing LPA analogs up-regulated the CD36 scavenger receptor in vitro and in vivo and elicited dedifferentiation of cultured vascular smooth muscle cells that was prevented by GW9662. These results suggest that selected LPA analogs are important novel endogenous PPARγ ligands capable of mediating vascular remodeling and that activation of the nuclear transcription factor PPARγ is possible necessary and sufficient for neointima formation by components of oxidized low d. lipoprotein.

AN 2004:242383 CAPLUS <<LOGINID::20080311>>
DN 140:373126
TI Lysophosphatidic acid induces neointima formation through PPARγ activation
AU Zhang, Chunxiang; Baker, Daniel L.; Yasuda, Satoshi; Makarova, Natalia; Balazs, Louisa; Johnson, Leonard R.; Marathe, Gopal K.; McIntyre, Thomas M.; Xu, Yong; Prestwich, Glenn D.; Byun, Hoe-Sup; Bittman, Robert; Tigyi, Gabor
CS Vascular Biology Center of Excellence, The University of Tennessee Health Science Center, Memphis, TN, 38163, USA
SO Journal of Experimental Medicine (2004), 199(6), 763-774
CODEN: JEMEAV; ISSN: 0022-1007
PB Rockefeller University Press
DT Journal
LA English
RE.CNT 53 THERE ARE 53 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L32 ANSWER 9 OF 9 CAPLUS COPYRIGHT 2008 ACS on STN
TI Athero- and thrombogenic actions of lysophosphatidic acid and sphingosine-1-phosphate
AB A review. Lysophosphatidic acid (LPA) and sphingosine-1-phosphate (S1P) are potent bioactive phospholipids with specific and multiple effects on blood cells and cells of the vessel wall. Released by activated platelets, LPA and S1P mediate physiol. wound healing processes such as vascular repair. Evidence is accumulating that these lipid mediators can, however, under certain conditions become athero- and thrombogenic mols. that might aggravate cardiovascular disease. For example, LPA present in minimally modified LDL and within the intima of atherosclerotic lesions may play a role in the early phase of atherosclerosis by inducing barrier dysfunction and increased monocyte adhesion of the endothelium, as well as in the late phase by triggering platelet activation and intra-arterial thrombus formation upon rupture of the atherosclerotic plaque. Moreover, LPA and S1P, by stimulating the proliferation of fibroblasts and by enhancing the survival of inflammatory cells are likely to play a central role in the excessive fibroproliferative and inflammatory response to vascular injury that characterizes the progression of atherosclerosis. Furthermore, LPA can cause the phenotypic dedifferentiation of medial vascular smooth muscle cells, and S1P is able to stimulate the migration and proliferation of intimal vascular smooth muscle cells; both processes ultimately lead to the formation of the neointima. Most importantly, as LPA and S1P bind to and activate multiple G-protein receptors, it emerges that the

beneficial or harmful action of LPA and S1P are critically dependent on the expression profile of their receptor subtypes and their coupling to different signal transduction pathways in the target cells. By targeting specific subtypes of LPA and S1P receptors in selective cells of the vascular wall and blood, new strategies for the prevention and therapy of cardiovascular diseases can be envisioned.
AN 2002:459264 CAPLUS <<LOGINID::20080311>>
DN 137:199092
TI Athero- and thrombogenic actions of lysophosphatidic acid and sphingosine-1-phosphate
AU Siess, Wolfgang
CS Medical Faculty, Institute for Prevention of Cardiovascular Diseases, University of Munich, Munich, D-80336, Germany
SO Biochimica et Biophysica Acta, Molecular and Cell Biology of Lipids (2002), 1582(1-3), 204-215
CODEN: BBMLFG; ISSN: 1388-1981
PB Elsevier B.V.
DT Journal; General Review
LA English
RE.CNT 160 THERE ARE 160 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> log hold
COST IN U.S. DOLLARS SINCE FILE ENTRY TOTAL SESSION
FULL ESTIMATED COST 37.27 172.94

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS) SINCE FILE ENTRY TOTAL SESSION
CA SUBSCRIBER PRICE -7.20 -33.60

SESSION WILL BE HELD FOR 120 MINUTES
STN INTERNATIONAL SESSION SUSPENDED AT 10:34:54 ON 11 MAR 2008

Connecting via Winsock to STN

Welcome to STN International! Enter x:x

LOGINID:SSPTAEXO1623

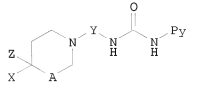
PASSWORD:
* * * * * * RECONNECTED TO STN INTERNATIONAL * * * * * *
SESSION RESUMED IN FILE 'CAPLUS' AT 10:41:56 ON 11 MAR 2008
FILE 'CAPLUS' ENTERED AT 10:41:56 ON 11 MAR 2008
COPYRIGHT (C) 2008 AMERICAN CHEMICAL SOCIETY (ACS)d

COST IN U.S. DOLLARS SINCE FILE ENTRY TOTAL SESSION
FULL ESTIMATED COST 37.27 172.94

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS) SINCE FILE ENTRY TOTAL SESSION
CA SUBSCRIBER PRICE -7.20 -33.60

=> d 117 1-15 ti abs bib
YOU HAVE REQUESTED DATA FROM FILE 'HCAPLUS' - CONTINUE? (Y)/N:y

L17 ANSWER 1 OF 15 HCAPLUS COPYRIGHT 2008 ACS on STN
TI Preparation of pyridinyl ureas as urotensin II antagonists
GI



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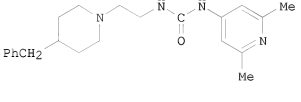

II

AB Title compds. I [wherein Py = pyridin-4-yl disubstituted in positions 2 and 6; X = aryl, arylalkyl, aryloxy, etc.; A = (CH2)n; XCZ form an exocyclic bond which bears an Ar group and the just formed CH2 group; Z = H; when X = aryl or arylalkyl, Z = H, OH, CO2H, etc.; when X = aryl, arylakyl and n = 0, Z = H, OH, CO2H, aryl, etc.; Y = CR6R7(CH2)m, (CH2)mCR6R7; m = 1-2; n = 0-1; R6 = H, alkyl, aryl, arylalkyl; or R6CR7 = carbocycle; R7 = H, Me; and their enantiomers, diastereomers, racemates, pharmaceutically acceptable salts, solvate complexes, and morphol. forms thereof] were prepared as neurohormonal antagonists. For example, reacting 2-(4-benzylpiperidino)-1-ethanamine with 1,3-Bis(2,6-dimethylpyridin-4-yl)urea gave II. In binding assays of human [125I]-urotensin II to human-derived TE-671 rhabdomyosarcoma cells, compds. of the invention showed activity with IC50 values ranging from 0.1 nM to 1000 nM. Thus, I and their pharmaceutical compns., optionally comprising other pharmacol. active compds., are useful for treating a variety of disorders associated with dysregulation of urotensin II, such as heart disease, hypertension, kidney disease, diabetes, asthma, and pulmonary disease (no data).

AN 2005:303504 HCAPLUS <<LOGINID::20080311>>
DN 142:355172

TI Preparation of pyridinyl ureas as urotensin II antagonists

IN Mathys, Boris; Mueller, Claus; Scherz, Michael; Weller, Thomas; Clozel, Martine; Velker, Joerg; Bur, Daniel
PA Actelion Pharmaceuticals Ltd., Switz.

SO PCT Int. Appl., 113 pp.
CODEN: PIXXD2
DT Patent
LA English

FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2005030209	A1	20050407	WO 2004-EP10559	20040921 <--

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC,

LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI,
NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY,
TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM,
AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK,
EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE,
SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE,
SN, TD, TG

AU	2004275488	A1	20050407	AU 2004-275488	20040921 <--
CA	2540196	A1	20050407	CA 2004-2540196	20040921 <--
EP	1670470	A1	20060621	EP 2004-765436	20040921 <--
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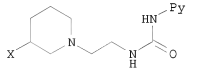
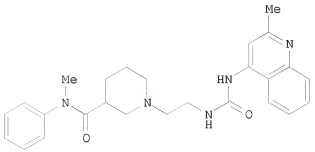
CN	1856305	A	20061101	CN 2004-80027725	20040921 <--
BR	2004014777	A	20061121	BR 2004-14777	20040921 <--
JP	2007506692	T	20070322	JP 2006-527332	20040921 <--
MX	2006PA03264	A	20060608	MX 2006-PA3264	20060323 <--
KR	2007014108	A	20070131	KR 2006-705848	20060324 <--
NO	2006001395	A	20060622	NO 2006-1395	20060327 <--
US	2007043081	A1	20070222	US 2006-573516	20060327 <--
IN	2006CN01415	A	20070622	IN 2006-CN1415	20060425 <--
PRAI WO	2003-EP10746	A	20030926	<--	
WO	2003-EP310746	A	20030926	<--	
WO	2004-EP10559	W	20040921		

OS MARPAT 142:355172
RE.CNT 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L17 ANSWER 2 OF 15 HCAPLUS COPYRIGHT 2008 ACS on STN
TI Preparation of novel piperidine derivatives as urotensin II antagonists
GI



I



II

AB The invention relates to novel piperidine derivs. I [Py = substituted pyridin-4-yl, (un)substituted quinolin-4-yl; X = CONR3R4; R1, R2 = H, alkyl, arylalkyl; R3, R4 = H, alkyl, aryl, etc.; or NR3R4 = pyrrolidine, piperidine, morpholine] and their use as as neurohormonal antagonists, in

particular their use as urotensin II antagonists. The multi-step synthesis of the urea II (no characterization data for intermediates), was provided. The compds. I were found to have IC50 values ranging from 10 to 1000 nM in the assay for evaluating inhibition of human [125I]-urotensin II binding to a rhabdomyosarcoma cell line. The pharmaceutical compns. containing one or more of those compds. I are disclosed.

AN 2004:996155 HCAPLUS <<LOGINID::20080311>>
DN 141:424119
TI Preparation of novel piperidine derivatives as urotensin II antagonists
IN Aissaoui, Hamed; Binkert, Christoph; Clozel, Martine; Mathys, Boris; Mueller, Claus; Nayler, Oliver; Scherz, Michael; Verker, Jorg; Weller, Thomas
PA Actelion Pharmaceuticals Ltd., Switz.
SO PCT Int. Appl., 32 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2004099180	A1	20041118	WO 2004-EP4717	20040504 <--
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	CA 2523568	A1	20041118	CA 2004-2523568	20040504 <--
	EP 1641776	A1	20060405	EP 2004-730993	20040504 <--
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK				
	CN 1784394	A	20060607	CN 2004-80012297	20040504 <--
	JP 2006525274	T	20061109	JP 2006-505366	20040504 <--
	US 2007010516	A1	20070111	US 2005-556029	20051108 <--
PRAI	WO 2003-EP4811	A	20030508	<--	
	WO 2003-EP304811	A	20030508	<--	
	WO 2004-EP4717	W	20040504		

OS MARPAT 141:424119
RE.CNT 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L17 ANSWER 3 OF 15 HCAPLUS COPYRIGHT 2008 ACS on STN
TI Lysophosphatidic acid analogs and inhibition of neointima formation
AB The phospholipid growth factor lysophosphatidic acids (LPAs) containing unsatd. fatty acids (18:1, 18:2 and 20:4) and fatty alcs. containing hydrocarbon chains with more than 4 carbons were capable of inducing a rapid formation of neointima, an initial step in the development of atherosclerotic plaque. LPAs with saturated fatty acids did not induce neointima formation. A Peroxisome Proliferator-Activated Receptors gamma (PPAR.gamma.)-specific agonist Rosiglitasone also induced a profound formation of neointima. GW9662, a selective and irreversible antagonist of PPAR.gamma., abolished LPA- and Rosiglitazone-induced neointima formation, indicating that LPA-induced neointima formation requires the activation of PPAR.gamma.. These data suggest that LPA analogs that bind to but

do not activate downstream signaling of PPAR.gamma. or antagonists of PPAR.gamma. that inhibit PPAR.gamma. signaling would be useful in the prevention and/or treatment of neointima formation and atherosclerosis.

AN 2004:857161 HCAPLUS <<LOGINID::20080311>>

DN 141:343506

TI Lysophosphatidic acid analogs and inhibition of neointima formation

IN Tigyi, Gabor; Baker, Daniel L.; Zhang, Chunxiang

PA USA

SO U.S. Pat. Appl. Publ., 23 pp.
CODEN: USXXCO

DT Patent

LA English

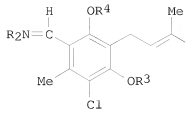
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 2004204383	A1	20041014	US 2004-821739	20040409 <--
	AU 2004229467	A1	20041028	AU 2004-229467	20040409 <--
	AU 2004229467	B2	20070125		
	CA 2521189	A1	20041028	CA 2004-2521189	20040409 <--
	WO 2004091496	A2	20041028	WO 2004-US11016	20040409 <--
	WO 2004091496	A3	20050324		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	EP 1613298	A2	20060111	EP 2004-759365	20040409 <--
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK, HR				
	JP 2007525449	T	20070906	JP 2006-509874	20040409 <--
PRAI	US 2003-462274P	P	20030411	<--	
	WO 2004-US11016	W	20040409		

L17 ANSWER 4 OF 15 HCAPLUS COPYRIGHT 2008 ACS on STN

TI Preparation of ascochlorin-amino acids Schiff bases or its analogs as novel transcriptional factor and process for producing the same and use thereof

GI



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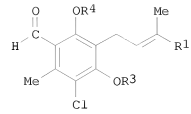
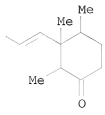
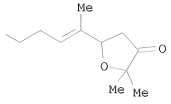


II

Q=



Q1=



AB Novel imino compds., i.e. 3-prenylbenzaldehyde-amino acid Schiff base (I) [R1 = Q, Q1; R2 = (CH2)nCHR5R6; n = 0-6; R5 = H, NH2, mono- or di(C1-6 alkyl)amino, phenyl-C1-6 alkyl; R6 = CO2H, CONH2, (un)substituted C1-6 alkoxycarbonyl; R3, R4 = H, each (un)substituted C1-6 alkyl, C2-6 alkenyl, C2-6 alkynyl, or C3-8 cycloalkyl, acyl, aryl, CO2H] are synthesized by mixing and reacting ascochlorin, its analogs or its derivs. (II; R1-R3 = same as above) with amino acids having a primary amino group of formula R5R6CH(CH2)nNH2 (R5, R6 = same as above) in the presence/absence of a basic catalyst. The novel imino compds. I thus synthesized are ligands which activate nuclear receptor superfamily such as retinoid orphan receptor (RXR), peroxisome proliferator activated receptor (PPAR) and steroid receptor (PXR) and show an effect of promoting the transcription of a drug-metabolizing enzyme CYP7A1. They have therapeutic effects on diseases such as life style-related diseases, diabetes, arteriosclerosis, multiple risk factor syndrome, myxedema, hypertension, or chronic inflammation. They are useful for the preventives and/or therapeutic agents for restenosis of arterial cavity enlarged by balloon catheter or stent or as serum cholesterol-lowering agents or adhesion promoters for adhering transplanted cells or tissues derived by differentiated induction of stem cells in a recipient. Thus, when a feed containing 0.025-0.1% compound (III) was fed to obese diabetic mice for 20 days, the excretion of sugar in urine was effectively reduced.

AN 2004:718503 HCAPLUS <<LOGINID::20080311>>
DN 141:225837

TI Preparation of ascochlorin-amino acids Schiff bases or its analogs as novel transcriptional factor and process for producing the same and use thereof

IN Kitahara, Takeshi; Watanabe, Hidenori; Ando, Kunio
PA NRL Pharma, Inc., Japan

SO PCT Int. Appl., 64 pp.
CODEN: PIXXD2
DT Patent
LA Japanese
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2004074236	A1	20040902	WO 2004-JP2110	20040224 <--

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH,

CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD,
GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC,
LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE,
BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU,
MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN,
GQ, GW, ML, MR, NE, SN, TD, TG

CA 2516698 A1 20040902 CA 2004-2516698 20040224 <--
EP 1616856 A1 20060118 EP 2004-714032 20040224 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK
US 20062473O7 A1 20061102 US 2005-546854 20050824
PRAI JP 2003-92682 A 20030224 <--
WO 2004-JP2110 W 20040224
OS MARPAT 141:225837
RE.CNT 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L17 ANSWER 5 OF 15 HCAPLUS COPYRIGHT 2008 ACS on STN
TI Endovascular implants especially stents that are coated with a combination of PPAR-agonists and RXR-agonists
AB The invention concerns endo-vascular implants especially stents that are coated at least partially with a combination of PPAR-agonists and RXR-agonists; the drugs can be incorporated in a carrier selected from the group of polylactide, poly-L-lactide or hyaluronic acid. The drugs are applied to treat and prevent stenosis and restenosis. Thus a com. stent (Lekton) was mounted onto a rotary atomizer; the fluid reservoir was filled with poly-l-lactide(Resomer L214) and clofibrate in chloroform. Coating was performed while the stent was rotated and the polylactide-clofibrate solution was sprayed periodically to allow time for solvent evaporation; both sides of the stent were sprayed in an 80 cycle process with 10 s spraying and 12 s dying. The stents were implanted in swine.
AN 2004:136474 HCAPLUS <<LOGINID::20080311>>
DN 140:169740
TI Endovascular implants especially stents that are coated with a combination of PPAR-agonists and RXR-agonists
IN Rohde, Roland; Sternberg, Katrin; Diener, Tobias
PA Biotronik Mess- und Therapiegeraete GmbH & Co. Ingenieurbuero Berlin, Germany
SO Eur. Pat. Appl., 11 pp.
CODEN: EPXXDW
DT Patent
LA German
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
EP 1389472	A2	20040218	EP 2003-90236	20030728 <--
EP 1389472	A3	20040421		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
DE 10237571	A1	20040226	DE 2002-10237571	20020813 <--
PRAI DE 2002-10237571	A	20020813 <--		

L17 ANSWER 6 OF 15 HCAPLUS COPYRIGHT 2008 ACS on STN
TI The association of Pro12Ala polymorphism in PPAR.gamma.2 with lower carotid artery IMT in Japanese
AB In this study, the association of the Pro12Ala peroxisome proliferator-activated receptor γ2 (PPAR.gamma.2) polymorphism with atherosclerosis was examined in a Japanese type 2 diabetic population. PPAR.gamma. has been

identified as a key regulator of adipogenesis. Recently, some studies reported that the Pro12Ala polymorphism was associated with resistance to Type 2 diabetes. It is well-known that Type 2 diabetes is closely related with disorder of lipid metabolism as well as impaired glucose homeostasis, resulting in atherosclerosis. We aimed to evaluate the association between carriers of the Pro12Ala PPAR.gamma.2 mutation and clin. profiles concerning atherosclerosis besides plasma glucose and lipid concns. Screening for the mutation was performed using the PCR -restriction fragment length polymorphism (PCR-RFLP) method among 154 type 2 diabetic patients. The homozygotes of the Pro12 allele were 143 (93%), the heterozygotes of the Pro12 and Ala12 allele were 11 (7%) and the homozygote of the Ala12 allele was not detected. The group with the Ala12 allele had a significantly lower value of carotid artery intima-media thickness (IMT) than that without it, although there was no difference between 2 groups in sex, age or other clin. variables the authors examined The Pro12Ala PPAR.gamma.2 polymorphism may be associated with carotid artery IMT values in type 2 diabetes mellitus.

AN 2003:850595 HCAPLUS <<LOGINID::20080311>>
DN 140:126350
TI The association of Pro12Ala polymorphism in PPAR.gamma.2 with lower carotid artery IMT in Japanese
AU Iwata, E.; Yamamoto, I.; Motomura, T.; Tsubakimori, S.; Nohnen, S.; Ohmoto, M.; Igarashi, T.; Azuma, J.
CS Graduate School of Pharmaceutical Sciences, Department of Clinical Evaluation of Medicines and Therapeutics, Osaka University, 1-6 Yamadaqoka, Suita, Osaka, 565-0871, Japan
SO Diabetes Research and Clinical Practice (2003), 62(1), 55-59
CODEN: DRCPE9; ISSN: 0168-8227
PB Elsevier Science B.V.
DT Journal
LA English
RE.CNT 10 THERE ARE 10 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L17 ANSWER 7 OF 15 HCAPLUS COPYRIGHT 2008 ACS on STN
TI Peroxisome proliferator-activated receptor gamma ligand eluting medical device
AB Implantable medical devices having an anti-restenotic coatings of peroxisome proliferator-activated receptor γ (PPAR.gamma.) agonists are disclosed. The anti-restenotic PPAR.gamma. ligands include thiazolidinedione compds. including ciglitazone. The anti-restenotic medial devices include stents, catheters, micro-particles, probes and vascular grafts. The medical devices can be coated using any method known in the art including compounding the thiazolidinedione with a biocompatible polymer prior to applying the coating. Addnl., medical devices having a coating comprising at least one thiazolidinedione in combination with at least one addnl. therapeutic agent, such as an antiplatelet, antifibrotic, or anti-inflammatory agent, are also described. For example, a stainless steel stent was coated using a drug/polymer system. Ciglitazone (250 mg) was dissolved in THF and 251.6 mg of polycaprolactone (PCL) was added and mixed until the PCL dissolved forming a drug/polymer solution The cleaned, dried stents were coated using either spraying techniques or dipped into the drug/polymer solution to achieve a final coating weight of between approx. 10 μg to 1 mg. Finally, the coated stents were dried in a vacuum oven at 50° over night.
AN 2002:671834 HCAPLUS <<LOGINID::20080311>>
DN 137:206601
TI Peroxisome proliferator-activated receptor gamma ligand eluting medical device

IN Carlyle, Wenda; Cheng, Peiwen; Cafferata, Robert L.
PA Medtronic Ave, Inc., USA
SO Eur. Pat. Appl., 21 pp.
CODEN: EPXXDW
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI EP 1236478	A1	20020904	EP 2002-251370	20020227 <--
EP 1236478	B1	20051026		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
US 2002127263	A1	20020912	US 2002-85539	20020226 <--
AT 307622	T	20051115	AT 2002-251370	20020227 <--
EP 1647289	A1	20060419	EP 2005-18140	20020227 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI, CY, TR				
PRAI US 2001-271898P	P	20010227 <--		
EP 2002-251370	A3	20020227 <--		

RE.CNT 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L17 ANSWER 8 OF 15 HCAPLUS COPYRIGHT 2008 ACS on STN
TI Intimal smooth muscle cells as a target for peroxisome proliferator-activated receptor-γ ligand therapy
AB Activation of the nuclear receptor/transcription factor, peroxisome proliferator-activated receptor γ (PPAR.gamma.), is a newly defined target for limiting vascular pathologies. PPAR.gamma. is expressed in human and animal models of vascular disease, with particularly high levels being present in the cells of the neointimal microenvironment. In the present study, we show that intimal smooth muscle cells in vitro contain higher amts. of functional PPAR.gamma. than medial smooth muscle cells. The PPAR γ ligand rosiglitazone more potently induced CD36 expression at low concns., and cell death by apoptosis at higher concns. in intimal compared with medial smooth muscle cells. Intimal smooth muscle cells also contained high levels of cyclooxygenase-2 protein, and released a more diverse and larger amount of eicosanoids on arachidonic acid stimulation. Furthermore, when exogenous arachidonic acid was added, PPAR reporter gene activation was induced in a cyclooxygenase inhibitor-sensitive manner, an effect that correlated with an increase in CD36 expression. In summary, intimal smooth muscle cells contain functionally higher levels of PPAR.gamma., PPAR.gamma. ligands have high- and low-potency targets in vascular smooth muscle cells, and cyclooxygenase can serve as a source of potential endogenous PPAR ligands. Intimal vascular smooth muscle cells therefore represent a potentially important target for the antiproliferative, and antiatherosclerotic actions of PPAR.gamma. ligands.
AN 2002:629069 HCAPLUS <<LOGINID::20080311>>
DN 138:198356
TI Intimal smooth muscle cells as a target for peroxisome proliferator-activated receptor-γ ligand therapy
AU Bishop-Bailey, David; Hla, Timothy; Warner, Timothy D.
CS Department of Cardiac, Vascular Research, William Harvey Research Institute, Barts and the London, Queen Mary University of London, London, EC1 M 6BQ, UK
SO Circulation Research (2002), 91(3), 210-217

CODEN: CIRUAL; ISSN: 0009-7330
PB Lippincott Williams & Wilkins
DT Journal
LA English
RE.CNT 52 THERE ARE 52 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L17 ANSWER 9 OF 15 HCAPLUS COPYRIGHT 2008 ACS on STN
TI PPAR.gamma. ligand inhibits osteopontin gene expression through interference with binding of nuclear factors to A/T-rich sequence in THP-1 cells
AB Peroxisome proliferator-activated receptor γ(PPAR.gamma.) is a member of the nuclear receptor superfamily that acts as a key player in adipocyte differentiation, glucose metabolism, and macrophage differentiation. Osteopontin (OPN) a component of extracellular matrix, is elevated during neointimal formation in the vessel wall and is synthesized by macrophages in atherosclerotic plaques. In the present study. we investigated the mol. mechanisms regulating OPN gene expression by PPAR.gamma. in THP-1 cells, a cell line derived from human monocytic leukemia cells. Northern and Western blot analyses showed that exposure of THP-1 cells to PMA (phorbol 12-myristate 13-acetate) increases OPN mRNA and protein levels in a time-dependent manner. PMA-induced OPN expression was significantly decreased by troglitazone (Tro) and other PPAR.gamma. ligands. Transient transfection assays of the human OPN promoter/luciferase construct showed that PPARγ represses OPN promoter activity, and the PPAR.gamma.-responsive region within the OPN promoter lies between -1000 and -970 relative to the transcription start site. Site-specific mutation anal. and electrophoretic mobility shift assays indicated that a homeobox-like A/T-rich sequence between -990 and 981, which functions as a binding site for PMA-induced nuclear factors other than PPAR.gamma., mediates the repression of OPN expression by Tro. Furthermore, concatenated A/T-rich sequences conferred the PPAR.gamma. responsiveness on the heterologous promoter. Taken together, these data suggest that PPAR.gamma. ligand inhibits OPN gene expression through the interference with the binding of nuclear factors to A/T-rich sequence in THP-1 cells.
AN 2002:162012 HCAPLUS <<LOGINID::20080311>>
DN 136:338695
TI PPAR.gamma. ligand inhibits osteopontin gene expression through interference with binding of nuclear factors to A/T-rich sequence in THP-1 cells
AU Oyama, Yuko; Akuzawa, Nobuhiro; Nagai, Ryozo; Kurabayashi, Masahiko
CS Second Department of Internal Medicine, Gunma University School of Medicine, Maebashi, 371-8511, Japan
SO Circulation Research (2002), 90(3), 348-355
CODEN: CIRUAL; ISSN: 0009-7330
PB Lippincott Williams & Wilkins
DT Journal
LA English
RE.CNT 34 THERE ARE 34 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L17 ANSWER 10 OF 15 HCAPLUS COPYRIGHT 2008 ACS on STN
TI Peroxisome proliferator-activated receptor γ inhibits transforming growth factor β-induced connective tissue growth factor expression in human aortic smooth muscle cells by interfering with Smad3
AB Activation of peroxisome proliferator-activated receptor γ (PPAR.gamma.) after balloon injury significantly inhibits VSMC proliferation and

neointima formation. However, the precise mechanisms of this inhibition have not been determined The authors hypothesized that activation of PPAR.gamma. in vascular injury could attenuate VSMC growth and matrix production during vascular lesion formation. Since connective tissue growth factor (CTGF) is a key factor regulating extracellular matrix production, abrogation of transforming growth factor β (TGF-β)-induced CTGF production by PPAR.gamma. activation may be one of the mechanisms through which PPAR.gamma. agonists inhibit neointima formation after vascular injury. In this study, the authors demonstrate that the PPAR.gamma. natural ligand (15-deoxyprostaglandin J2) and a synthetic ligand (GW7845) significantly inhibit TGF-β-induced CTGF production in a dose-dependent manner in HASMCs. In addition, suppression of CTGF mRNA expression is relieved by pretreatment with an antagonist of PPAR.gamma. (GW9662), suggesting that the inhibition of CTGF expression is mediated by PPAR.gamma.. To elucidate further the mol. mechanism by which PPAR.gamma. inhibits CTGF expression, an .apprx.2-kilobase pair CTGF promoter was cloned. The authors found that PPAR.gamma. activation inhibits TGF-β-induced CTGF promoter activity in a dose-dependent manner, and suppression of CTGF promoter activity by PPAR.gamma. activation is completely rescued by overexpression of Smad3, but not by Smad4. Furthermore, PPAR.gamma. phys. interacts with Smad3 but not Smad4 in vitro in glutathione S-transferase pull-down expts. Taken together, the data suggest that PPAR γ inhibits TGF-β-induced CTGF expression in HASMCs by directly interfering with the Smad3 signaling pathway.

AN 2001:908512 HCAPLUS <<LOGINID::20080311>>

DN 136:198017

TI Peroxisome proliferator-activated receptor γ inhibits transforming growth factor β-induced connective tissue growth factor expression in human aortic smooth muscle cells by interfering with Smad3

AU Fu, Mingui; Zhang, Jifeng; Zhu, Xiaojun; Myles, David E.; Willson, Timothy M.; Liu, Xuedong; Chen, Yuqing E.

CS Cardiovascular Research Institute, Morehouse School of Medicine, Atlanta, GA, 30310, USA

SO Journal of Biological Chemistry (2001), 276(49), 45888-45894
CODEN: JBCHA3; ISSN: 0021-9258

PB American Society for Biochemistry and Molecular Biology

DT Journal

LA English

RE.CNT 30 THERE ARE 30 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L17 ANSWER 11 OF 15 HCAPLUS COPYRIGHT 2008 ACS on STN

TI Pioglitazone enhances cytokine-induced apoptosis in vascular smooth muscle cells and reduces intimal hyperplasia

AB Cytokines induce apoptosis in vascular disease lesions through enhancement of inducible NO synthase (iNOS) activation. The thiazolidinediones, novel insulin-sensitizing agents, were demonstrated to modulate cytokine-induced NO production The authors have investigated the role of pioglitazone in the apoptosis of vascular smooth muscle cells (VSMCs) in vitro and developed intimal hyperplasia in vivo. Pioglitazone (0.1 to 10 μmol/L) significantly enhanced cytokine-induced expression of iNOS and NO production in a dose-dependent manner in rat VSMCs, but 15-deoxy-Δ12,14-prostaglandin J2 (≤ 10 μmol/L), a native peroxisome proliferator-activated receptor-γ ligand, showed no effect. Pioglitazone also significantly enhanced reduction of cell viability, as evidenced by the increase in the number of TUNEL-pos. cells. All of these effects of pioglitazone were blocked by treatment with N-monomethyl-L-Arg, an NO synthesis inhibitor. In an in vivo study

with a balloon-injured rat carotid artery, neointimal thickness had reached maximum levels at 2 wk after injury. Then, rats were fed with or without pioglitazone (3 mg · kg-1 · d-1) for an addnl. week. The ratio of intima to media area of carotid artery was significantly decreased by 30%, and the ratio of apoptotic cells in neointima was significantly increased in pioglitazone-treated rats compared with vehicle-treated control rats. Pioglitazone enhanced apoptosis in an NO-dependent manner in cytokine-activated VSMCs and induced significant regression of intimal hyperplasia in balloon-injured rat carotid artery. It appears that pioglitazone is a potent apoptosis inducer in vascular lesions, providing a novel pharmacol. strategy to prevent restenosis after vascular intervention.

AN 2001:613879 HCAPLUS <<LOGINID::20080311>>
DN 136:303789
TI Pioglitazone enhances cytokine-induced apoptosis in vascular smooth muscle cells and reduces intimal hyperplasia
AU Aizawa, Yoshiaki; Kawabe, Jun-ichi; Hasebe, Naoyuki; Takehara, Naohumi; Kikuchi, Kenjiro
CS Department of Medicine, Asahikawa Medical College, Asahikawa, Japan
SO Circulation (2001), 104(4), 455-460
CODEN: CIRCAZ; ISSN: 0009-7322
PB Lippincott Williams & Wilkins
DT Journal
LA English
RE.CNT 29 THERE ARE 29 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L17 ANSWER 12 OF 15 HCAPLUS COPYRIGHT 2008 ACS on STN
TI Control of vascular cell proliferation and migration by PPAR-γ: A new approach to the macrovascular complications of diabetes
AB A review with 82 refs. Compared with nondiabetic subjects, type 2 diabetic individuals are at an increased risk for coronary artery disease and coronary restenosis after angioplasty or stenting. Increased proliferation and migration of vascular smooth muscle cells (VSMCs) contribute importantly to the formation of both atherosclerotic and restenotic lesions. Therefore, pharmaceutical interventions targeting proteins that regulate VSMC growth or movement are a promising new approach to treat diabetes-associated cardiovascular disease. Peroxisome proliferator-activated receptor-γ (PPAR-γ) is a member of the nuclear receptor superfamily that, when activated by thiazolidinedione (TZD) insulin sensitizers, regulates a host of target genes. All of the major cells in the vasculature express PPAR-γ, including endothelial cells, VSMCs, and monocytes/macrophages. PPAR-γ is present in intimal macrophages and VSMCs in early human atheromas. In an animal model of vascular injury, PPAR-γ levels are substantially elevated in the neointima that forms after mech. injury of the endothelium. Recent exptl. studies provide evidence that PPAR-γ may function to protect the vasculature from injury. Cell culture studies have shown that TZD PPAR-γ ligands inhibit both the proliferation and migration of VSMCs. These antiatherogenic activities of PPAR-γ may also occur in vivo, because TZDs inhibit lesion formation in several animal models. PPAR-γ ligands may also protect the vasculature indirectly by normalizing metabolic abnormalities of the diabetic milieu that increase cardiovascular risk. Activation of PPAR-γ, newly defined in vascular cells, may be a useful approach to protect the vasculature in diabetes.

AN 2001:136312 HCAPLUS <<LOGINID::20080311>>
DN 134:235155
TI Control of vascular cell proliferation and migration by PPAR

-γ: A new approach to the macrovascular complications of diabetes
AU Hsueh, Willa A.; Jackson, Simon; Law, Ronald E.
CS Department of Medicine, the Endocrinology, Diabetes, and Hypertension Division, University of California School of Medicine, Los Angeles, CA, 90095-7073, USA
SO Diabetes Care (2001), 24(2), 392-397
CODEN: DICAD2; ISSN: 0149-5992
PB American Diabetes Association, Inc.
DT Journal; General Review
LA English
RE.CNT 82 THERE ARE 82 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L17 ANSWER 13 OF 15 HCAPLUS COPYRIGHT 2008 ACS on STN
TI Peroxisome proliferator-activated receptor γ activators downregulate angiotensin II type 1 receptor in vascular smooth muscle cells
AB Peroxisome proliferator-activated receptor γ (PPAR.gamma.) activators, such as troglitazone (Tro), not only improve insulin resistance but also suppress the neointimal formation after balloon injury. However, the precise mechanisms have not been determined Angiotensin II (Ang II) plays crucial roles in the pathogenesis of atherosclerosis, hypertension, and neointimal formation after angioplasty. The authors examined the effect of PPAR.gamma. activators on the expression of Ang II type 1 receptor (AT1-R) in cultured vascular smooth muscle cells (VSMCs). AT1-R mRNA and AT1-R protein levels were determined by Northern blot anal. and radioligand binding assay, resp. Natural PPAR.gamma. ligand 15-deoxy-Δ12.14-prostaglandin J2, as well as Tro, reduced the AT1-R mRNA expression and the AT1-R protein level. The PPAR.gamma. activators also reduced the calcium response of VSMCs to Ang II. PPAR.gamma. activators suppressed the AT1-R promoter activity measured by luciferase assay but did not affect the AT1-R mRNA stability, suggesting that the suppression occurs at the transcriptional level. PPAR.gamma. activators reduced the AT1-R expression and calcium response to Ang II in VSMCs. Downregulation of AT1-R may contribute to the inhibition of neointimal formation by PPAR.gamma. activators.
AN 2000:759543 HCAPLUS <<LOGINID::20080311>>
DN 134:66617
TI Peroxisome proliferator-activated receptor γ activators downregulate angiotensin II type 1 receptor in vascular smooth muscle cells
AU Takeda, Kotaro; Ichiki, Toshihiro; Tokunou, Tomotake; Funakoshi, Yuko; Iino, Naoko; Hirano, Katsuya; Kanaide, Hideo; Takeshita, Akira
CS Departments of Cardiovascular Medicine, Kyushu University Graduate School of Medical Sciences, Fukuoka, 812-8582, Japan
SO Circulation (2000), 102(15), 1834-1839
CODEN: CIRCAZ; ISSN: 0009-7322
PB Lippincott Williams & Wilkins
DT Journal
LA English
RE.CNT 26 THERE ARE 26 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L17 ANSWER 14 OF 15 HCAPLUS COPYRIGHT 2008 ACS on STN
TI Expression and function of PPAR.gamma. in rat and human vascular smooth muscle cells
AB Peroxisome proliferator-activated receptor-γ (PPAR.gamma.) is activated by fatty acids, eicosanoids, and insulin-sensitizing thiazolidinediones (TZDs).

The TZD troglitazone (TRO) inhibits vascular smooth muscle cell (VSMC) proliferation and migration in vitro and in post-injury intimal hyperplasia. Rat and human VSMCs expressed mRNA and nuclear PPAR γ1. Three PPAR.gamma. ligands, the TZDs TRO and rosiglitazone and the prostanoid 15-deoxy-Δ12,14-prostaglandin J2 (15d-PGJ2), all inhibited VSMC proliferation and migration. PPAR γ was upregulated in rat neointima at 7 days and 14 days after balloon injury and was also present in early human atheroma and precursor lesions. Thus, pharmacol. activation of PPAR.gamma. expressed in VSMCs inhibits their proliferation and migration, potentially limiting restenosis and atherosclerosis. These receptors are upregulated during vascular injury.

AN 2000:240919 HCAPLUS <<LOGINID::20080311>>
DN 133:148479
TI Expression and function of PPAR.gamma. in rat and human vascular smooth muscle cells
AU Law, Ronald E.; Goetze, Stephan; Xi, Xiao-Ping; Jackson, Simon; Kawano, Yasuko; Demer, Linda; Fishbein, Michael C.; Meehan, Woerner P.; Hsueh, Wilia A.
CS Department of Medicine, University of California at Los Angeles School of Medicine, Los Angeles, CA, 90095, USA
SO Circulation (2000), 101(11), 1311-1318
CODEN: CIRCAZ; ISSN: 0009-7322
PB Lippincott Williams & Wilkins
DT Journal
LA English
RE.CNT 33 THERE ARE 33 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L17 ANSWER 15 OF 15 HCAPLUS COPYRIGHT 2008 ACS on STN
TI A systematic analysis of 40 random genes in cultured vascular smooth muscle subtypes reveals a heterogeneity of gene expression and identifies the tight junction gene zonula occludens 2 as a marker of epithelioid "pup" smooth muscle cells and a participant in carotid neointimal formation
AB An accumulation of evidence suggests that vascular smooth muscle is composed of cell subpopulations with distinct patterns of gene expression. Much of this evidence has come from serendipitous discoveries of genes marking phenotypically distinct aortic cultures derived from 12-day-old and 3-mo-old rats. To identify more systematic differences, we isolated 40 genes at random from libraries of these 2 cultures and examined message expression patterns. To determine consistency of differential expression, we measured mRNA levels in 4 sets of cultures in 6 phenotypically distinct aortic cell clones and in balloon injured rat carotid arteries to determine the relevance of these differences in vitro to in vivo biol. The following 5 consistently differentially expressed genes were identified in vitro: zonula occludens 2 (ZO-2); peroxisome proliferator-activated receptor δ (PPAR.delta.); secreted protein, acidic and rich in cysteine (SPARC); α1(I)collagen; and A2, an uncharacterized gene. We examined these 5 clones during carotid artery injury and an inconsistently differentially expressed clone Krox-24 because, as an early response transcription factor, it could be involved in the injury response. PPAR δ, A2, and Krox-24 mRNAs were upregulated during the day after injury. ZO-2 and α1(I)collagen messages were modulated for up to a month, whereas SPARC message showed no consistent change. An anal. of ZO-2 and other tight junction genes indicates that tight junctions may play a role in smooth muscle biol. These data suggest that a systematic anal. of these libraries is likely to identify a very large number of differentially expressed genes. ZO-2 is particularly intriguing both because of this tight junction gene's pattern of prolonged over-expression

after injury and because of its potential role in determining the distinctive epithelioid phenotype of smooth muscle cells identified in rat and other species.
AN 1999:782811 HCAPLUS <<LOGINID::20080311>>
DN 132:289502
TI A systematic analysis of 40 random genes in cultured vascular smooth muscle subtypes reveals a heterogeneity of gene expression and identifies the tight junction gene zonula occludens 2 as a marker of epithelioid "pup" smooth muscle cells and a participant in carotid neointimal formation
AU Adams, Lawrence D.; Lemire, Joan M.; Schwartz, Stephen M.
CS Department of Pathology, University of Washington, Seattle, WA, 98195-7335, USA
SO Arteriosclerosis, Thrombosis, and Vascular Biology (1999), 19(11), 2600-2608
CODEN: ATVBFA; ISSN: 1079-5642
PB Lippincott Williams & Wilkins
DT Journal
LA English
RE.CNT 56 THERE ARE 56 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> log hold

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.48	219.76
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STN INTERNATIONAL SESSION SUSPENDED AT 10:42:07 ON 11 MAR 2008

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PASSWORD:
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COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION

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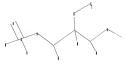

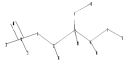

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chain bonds :
1-2 1-4 1-6 1-14 2-3 2-13 3-9 4-5 4-15 5-7 6-8 9-10 9-11 9-12
exact/norm bonds :
1-6 2-3 3-9 4-5 5-7 6-8 9-10 9-11 9-12
exact bonds :
1-2 1-4 1-14 2-13 4-15

G1:C,H,P

Match level :
1:CLASS 2:CLASS 3:CLASS 4:CLASS 5:CLASS 6:CLASS 7:CLASS 8:CLASS 9:CLASS
10:CLASS 11:CLASS 12:CLASS 13:CLASS 14:CLASS 15:CLASS

L33 STRUCTURE UPLOADED

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=> s l33
SAMPLE SEARCH INITIATED 11:05:14 FILE 'REGISTRY'
SAMPLE SCREEN SEARCH COMPLETED -     1390 TO ITERATE

100.0% PROCESSED     1390 ITERATIONS                                  50 ANSWERS
INCOMPLETE SEARCH (SYSTEM LIMIT EXCEEDED)
SEARCH TIME: 00.00.01

FULL FILE PROJECTIONS:  ONLINE   **COMPLETE**
                        BATCH    **COMPLETE**
PROJECTED ITERATIONS:          25564 TO    30036
PROJECTED ANSWERS:             18904 TO    22776

L34          50 SEA SSS SAM L33

=> d l34 scan

L34  50 ANSWERS   REGISTRY  COPYRIGHT 2008 ACS on STN
IN   D-myo-Inositol, 2,6-bis-O-(phenylmethyl)-, 1-[(2R)-2,3-bis[(1-
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     phosphate]
MF   C88 H104 O22 P4

Absolute stereochemistry.
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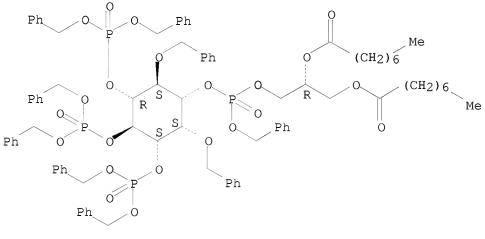

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**PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT**


HOW MANY MORE ANSWERS DO YOU WISH TO SCAN? (1):1

L34  50 ANSWERS   REGISTRY  COPYRIGHT 2008 ACS on STN
IN   Eicosapentaenoic acid, (1R)-1-[[[(2-aminoethoxy)hydroxyphosphinyl]oxy]meth
     yl]-2-[(1-oxoeicosyl)oxy]ethyl ester, (Z,Z,Z,Z,Z)-
MF   C45 H80 N O8 P
CI   IDS

     CM   1

Absolute stereochemistry.
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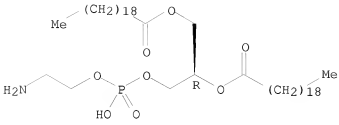

PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

HOW MANY MORE ANSWERS DO YOU WISH TO SCAN? (1):0

=> s l33 sss full
FULL SEARCH INITIATED 11:05:35 FILE 'REGISTRY'
FULL SCREEN SEARCH COMPLETED - 27926 TO ITERATE

100.0% PROCESSED 27926 ITERATIONS 21166 ANSWERS
SEARCH TIME: 00.00.01

L35 21166 SEA SSS FUL L33

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	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	178.36	398.12

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=> s l35/thu
36417 L35

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        987072 THU/RL
L36       6231 L35/THU
                 (L35 (L) THU/RL)

=> s l2 and l36
         10685 PPAR
         20400 PEROXISOME
         13861 PROLIFERATOR
        551870 ACTIVATED
        742262 RECEPTOR
          8211 PEROXISOME PROLIFERATOR-ACTIVATED RECEPTOR
                 (PEROXISOME(W)PROLIFERATOR(W)ACTIVATED(W)RECEPTOR)
L37          4 L2 AND L36

=> s l8 and l36
          1938 NEOINTIMA
          9253 RESTENOSIS
          5656 STENT
L38         54 L8 AND L36

=> s l38 and (PY<2004 or AY<2004 or PRY<2004)
      23979567 PY<2004
       4765121 AY<2004
       4243738 PRY<2004
L39         44 L38 AND (PY<2004 OR AY<2004 OR PRY<2004)

=> d l37 1-4 ti abs bib

L37  ANSWER 1 OF 4  CAPLUS  COPYRIGHT 2008 ACS on STN
TI   Linoleic Acid-Enriched Phospholipids Act through Peroxisome
     Proliferator-Activated Receptors α To Stimulate Hepatic
     Apolipoprotein A-I Secretion
AB   A uniquely formulated soy phospholipid, phosphatidylinositol (PI), is
     under development as a therapeutic agent for increasing plasma high-d.
     lipoprotein (HDL) levels.  Soy PI has been shown to increase plasma HDL
     and apolipoprotein A-I (apoA-I) levels in phase I human trials.  Low
     micromolar concns. of PI increase the secretion of apoA-I in model human
     hepatoma cell lines, through activation of G-protein and mitogen-activated
     protein (MAP) kinase pathways.  Expts. were undertaken to determine the
     importance of the PI head group and acyl chain composition on hepatic apoA-I
     secretion.  Phospholipids with choline and inositol head groups and one or
     more linoleic acid (LA) acyl chains were shown to stimulate apoA-I
     secretion by HepG2 cells and primary human hepatocytes.  Phospholipids
     containing two LA groups (dilinoleoylphosphatidylcholine, DLPC) were twice as
     active as those with only one LA group and promoted a 4-fold stimulation
     in apoA-I secretion.  Inhibition of cytosolic phospholipase A2 with
     pyrrolidine 1 (10 μM) resulted in complete attenuation of PI- and
     DLPC-induced apoA-I secretion.  Pretreatment with the peroxisome
     proliferator-activated receptor α (
     PPAR.alpha.) inhibitor MK886 (10 μM) also completely blocked
     PI- and DLPC-induced apoA-I secretion.  Hepatic PPAR.alpha.
     expression was significantly increased by both PI and DLPC.  However, in
     contrast to that seen with the fibrate drugs, PI caused minimal inhibition
     of catalytic activities of cytochrome P 450 and UGT1A1 enzymes.  These
     data suggest that LA-enriched phospholipids stimulate hepatic apoA-I
     secretion through a MAP kinase stimulation of PPAR.alpha..
     LA-enriched phospholipids have a greater apoA-I secretory activity than
     the fibrate drugs and a reduced likelihood to interfere with concomitant
     drug therapies.
AN   2008:58022  CAPLUS <<LOGINID::20080311>>
DN   148:229144
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TI Linoleic Acid-Enriched Phospholipids Act through Peroxisome Proliferator-Activated Receptors α To Stimulate Hepatic Apolipoprotein A-I Secretion
AU Pandey, Nihar R.; Renwick, Joanna; Misquith, Ayesha; Sokoll, Ken; Sparks, Daniel L.
CS Liponex, Inc., Ottawa, ON, K2G 3R8, Can.
SO Biochemistry (2008), 47(6), 1579-1587
CODEN: BICHAW; ISSN: 0006-2960
PB American Chemical Society
DT Journal
LA English
RE.CNT 64 THERE ARE 64 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L37 ANSWER 2 OF 4 CAPLUS COPYRIGHT 2008 ACS on STN
TI Method and compound for the treatment of valvular stenosis using a reverse lipid transport agonist
AB A method for treating valvular stenosis The method involves the administration of a therapeutically effective amount of a reverse lipid (in particular cholesterol) transport agonist to a mammal. The reverse lipid transport agonist is selected from the group consisting of a high d. lipoprotein (HDL), a peptide with HDL-like physiol. effects, a peptide with HDL-like physiol. effects complexed to a lipid, an HDL-mimetic agent, a cholesteryl ester transfer protein (CETP) modulator, a scavenger receptor class B, member 1 (SRB1) modulator, a liver X receptor/retinoid X receptor (LXR/RXR) agonist, an ATP-binding cassette transporter-1 (ABCA1) agonist and a peroxisome proliferator-
activated receptor (PPAR) agonist. Most
preferred is an apolipoprotein A-1 mimetic peptide/phospholipid complex.
AN 2007:1396181 CAPLUS <<LOGINID::20080311>>
DN 148:24443
TI Method and compound for the treatment of valvular stenosis using a reverse lipid transport agonist
IN Tardif, Jean-Claude
PA Institut de Cardiologie de Montreal, Can.
SO PCT Int. Appl., 45pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2007137400	A1	20071206	WO 2007-CA895	20070523
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BH, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW			
	RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, MT, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM			
PRAI US 2006-809850P		P	20060601		

RE.CNT 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L37 ANSWER 3 OF 4 CAPLUS COPYRIGHT 2008 ACS on STN
TI Pharmacological method for treatment of neuropathic pain

AB Disclosed are methods and compns. useful for treatment of neuropathic pain. In particular, the present invention provides methods of activating gamma-subtype peroxisome proliferator-activated receptors (PPAR γ) to inhibit, relieve, or treat neuropathic pain.
AN 2007:1213033 CAPLUS <<LOGINID::20080311>>
DN 147:480401
TI Pharmacological method for treatment of neuropathic pain
IN Taylor, Bradley K.
PA USA
SO U.S. Pat. Appl. Publ., 24pp.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 2007249561	A1	20071025	US 2007-739811	20070425
	WO 2007127791	A2	20071108	WO 2007-US67406	20070425
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	RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, MT, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM			
PRAI	US 2006-795078P	P	20060425		

OS MARPAT 147:480401

L37 ANSWER 4 OF 4 CAPLUS COPYRIGHT 2008 ACS on STN
TI Lysophosphatidic acid reduces the organ injury caused by endotoxemia-A role for G-protein-coupled receptors and peroxisome proliferator-activated receptor-γ
AB Exogenous lysophosphatidic acid (LPA) has been shown to beneficial in renal ischemia/reperfusion injury, wound healing and colitis. LPA acts via specific G-protein-coupled receptors and also peroxisome proliferator-activated receptor-γ (PPAR-γ). However, activation of PPAR-γ is dependent on the presence of an unsatd. acyl chain. Here we investigate the effects of saturated LPA (18:0) and unsatd. LPA (18:1) on the organ injury associated with endotoxemia and the receptors mediating LPA activity. Male Wistar rats received either lipopolysaccharide (LPS, 6 mg/kg i.v.) or vehicle. The PPAR-γ antagonist GW9662 (1 mg/kg i.v.), the LPA receptor antagonist Ki16425 (0.5 mg/kg i.v.) or vehicle was administered 30 min after LPS. LPA 18:0 or LPA 18:1 (1 mg/kg i.v.) or vehicle was administered 1 h after injection of LPS. Endotoxemia for 6 h resulted in an increase in serum levels of aspartate aminotransferase, alanine aminotransferase and creatine kinase. Therapeutic administration of LPA 18:0 or 18:1 reduced the organ injury caused by LPS. LPA 18:0 also attenuated the increase in plasma IL-1β caused by LPS. Ki16425, but not GW9662, attenuated the beneficial effects of LPA 18:0, however, Ki16425 and GW9662 attenuated the beneficial effects of 18:1. In conclusion, LPA reduces the organ injury caused by endotoxemia in the rat. Thus, LPA may be useful in the treatment of shock of various etiologies. The mechanism of action is related to acyl chain saturation, with LPA 18:0 acting via G-protein-coupled receptors and LPA 18:1 acting via G-protein-coupled receptors and PPAR-γ.

AN 2007:119866 CAPLUS <<LOGINID::20080311>>
DN 146:266187
TI Lysophosphatidic acid reduces the organ injury caused by endotoxemia-A role for G-protein-coupled receptors and peroxisome proliferator-activated receptor-γ
AU Murch, Oliver; Collin, Marika; Thiemermann, Christoph
CS Centre for Experimental Medicine, Nephrology & Critical Care, The William Harvey Research Institute, St. Bartholomew's and The Royal London School of Medicine and Dentistry, Queen Mary, University of London, London, EC1M 6BQ, UK
SO Shock (2007), 27(1), 48-54
CODEN: SAGUAI; ISSN: 1073-2322
PB Lippincott Williams & Wilkins
DT Journal
LA English
RE.CNT 32 THERE ARE 32 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> d l39 1-44 ti

L39 ANSWER 1 OF 44 CAPLUS COPYRIGHT 2008 ACS on STN
TI Endothelial cell specifically binding peptides and their use for targeting of gene delivery vectors

L39 ANSWER 2 OF 44 CAPLUS COPYRIGHT 2008 ACS on STN
TI Oxidized lipids and uses thereof in the treatment of inflammatory diseases and disorders

L39 ANSWER 3 OF 44 CAPLUS COPYRIGHT 2008 ACS on STN
TI Use of lipid conjugates in the treatment of diseases

L39 ANSWER 4 OF 44 CAPLUS COPYRIGHT 2008 ACS on STN
TI Methods and compounds for the treatment of vascular stenosis using a combination of N-phenyl-2-pyrimidine derivatives and PI3K inhibitors

L39 ANSWER 5 OF 44 CAPLUS COPYRIGHT 2008 ACS on STN
TI P-selectin targeting compositions containing P-selectin targeting peptides conjugated with lipids

L39 ANSWER 6 OF 44 CAPLUS COPYRIGHT 2008 ACS on STN
TI Method and system for systemic delivery of growth arresting, lipid-derived bioactive compounds

L39 ANSWER 7 OF 44 CAPLUS COPYRIGHT 2008 ACS on STN
TI Human p53 deletion mutant proteins and therapeutic use in cancer therapy

L39 ANSWER 8 OF 44 CAPLUS COPYRIGHT 2008 ACS on STN
TI Methods and compositions using defined oxidized phospholipids for prevention and treatment of atherosclerosis and other disorders

L39 ANSWER 9 OF 44 CAPLUS COPYRIGHT 2008 ACS on STN
TI Method using apolipoprotein-sphingomyelin complexes for treatment of dyslipidemic disorders

L39 ANSWER 10 OF 44 CAPLUS COPYRIGHT 2008 ACS on STN
TI Peptide and peptide analog apolipoprotein A-I agonists and their use to treat dyslipidemic disorders

L39 ANSWER 11 OF 44 CAPLUS COPYRIGHT 2008 ACS on STN
TI Compositions and methods for dosing liposomes of certain sizes to treat or

prevent disease

L39 ANSWER 12 OF 44 CAPLUS COPYRIGHT 2008 ACS on STN
TI Integrin targeted imaging agents

L39 ANSWER 13 OF 44 CAPLUS COPYRIGHT 2008 ACS on STN
TI Intramural Delivery of Recombinant Apolipoprotein A-IMilano/Phospholipid Complex (ETC-216) Inhibits In-Stent Stenosis in Porcine Coronary Arteries

L39 ANSWER 14 OF 44 CAPLUS COPYRIGHT 2008 ACS on STN
TI Drug delivery device with protective separating layer

L39 ANSWER 15 OF 44 CAPLUS COPYRIGHT 2008 ACS on STN
TI Peptide and peptide analog apolipoprotein A-I agonists, and their use to treat dyslipidemic disorders

L39 ANSWER 16 OF 44 CAPLUS COPYRIGHT 2008 ACS on STN
TI Gene transfer of human vascular endothelial growth factor 165 for prevention of stent restenosis after transjugular intrahepatic portosystemic shunt

L39 ANSWER 17 OF 44 CAPLUS COPYRIGHT 2008 ACS on STN
TI Therapy of proliferative disorders by direct irradiation of cell nuclei with tritiated nuclear targeting agents

L39 ANSWER 18 OF 44 CAPLUS COPYRIGHT 2008 ACS on STN
TI Preparation of prodrugs of 3-(pyrrol-2-ylmethylidene)-2-indolinones and activity as modulators of protein kinases

L39 ANSWER 19 OF 44 CAPLUS COPYRIGHT 2008 ACS on STN
TI Methods employing and compositions containing defined oxidized phospholipids for prevention and treatment of atherosclerosis

L39 ANSWER 20 OF 44 CAPLUS COPYRIGHT 2008 ACS on STN
TI Receptor antagonist-lipid conjugates and delivery vehicles containing same

L39 ANSWER 21 OF 44 CAPLUS COPYRIGHT 2008 ACS on STN
TI Cationic lipid-mediated transfection of bovine aortic endothelial cells inhibits their attachment

L39 ANSWER 22 OF 44 CAPLUS COPYRIGHT 2008 ACS on STN
TI Optimization of nonviral gene transfer of vascular smooth muscle cells in vitro and in vivo

L39 ANSWER 23 OF 44 CAPLUS COPYRIGHT 2008 ACS on STN
TI Surface modification of liposomes for selective cell targeting in cardiovascular drug delivery

L39 ANSWER 24 OF 44 CAPLUS COPYRIGHT 2008 ACS on STN
TI Focal arterial transgene expression after local gene delivery

L39 ANSWER 25 OF 44 CAPLUS COPYRIGHT 2008 ACS on STN
TI Pharmaceutical composition in the form of a nucleic acid lipid complex, the production thereof and its use in gene therapy

L39 ANSWER 26 OF 44 CAPLUS COPYRIGHT 2008 ACS on STN
TI Implantable depot drug delivery systems

L39 ANSWER 27 OF 44 CAPLUS COPYRIGHT 2008 ACS on STN
TI Toxicity, uptake kinetics and efficacy of new transfection reagents:

Increase of oligonucleotide uptake

L39 ANSWER 28 OF 44 CAPLUS COPYRIGHT 2008 ACS on STN
TI Preparation of vitronectin receptor antagonist pharmaceuticals

L39 ANSWER 29 OF 44 CAPLUS COPYRIGHT 2008 ACS on STN
TI Preparation of vitronectin receptor antagonist pharmaceuticals

L39 ANSWER 30 OF 44 CAPLUS COPYRIGHT 2008 ACS on STN
TI Preparation of vitronectin receptor antagonist pharmaceuticals

L39 ANSWER 31 OF 44 CAPLUS COPYRIGHT 2008 ACS on STN
TI Ribozyme therapy for the treatment and/or prevention of restenosis

L39 ANSWER 32 OF 44 CAPLUS COPYRIGHT 2008 ACS on STN
TI Agent for gene therapy of tumors and neurodegenerative, cardiovascular, and autoimmune diseases

L39 ANSWER 33 OF 44 CAPLUS COPYRIGHT 2008 ACS on STN
TI Ribozyme-mediated inhibition of cell proliferation: A model for identifying and refining a therapeutic ribozyme

L39 ANSWER 34 OF 44 CAPLUS COPYRIGHT 2008 ACS on STN
TI P-selectin translocation to vascular epithelial lumen by ionizing radiation, and therapeutic use

L39 ANSWER 35 OF 44 CAPLUS COPYRIGHT 2008 ACS on STN
TI Arterial Uptake of Biodegradable Nanoparticles: Effect of Surface Modifications

L39 ANSWER 36 OF 44 CAPLUS COPYRIGHT 2008 ACS on STN
TI Induction of E-selectin for targeting therapeutic agents to disease-associated vascular endothelial cells

L39 ANSWER 37 OF 44 CAPLUS COPYRIGHT 2008 ACS on STN
TI Lipid constructs for targeting oligonucleotides to vascular smooth muscle tissue

L39 ANSWER 38 OF 44 CAPLUS COPYRIGHT 2008 ACS on STN
TI Lipid constructs for cytoplasmic delivery of antisense oligonucleotides

L39 ANSWER 39 OF 44 CAPLUS COPYRIGHT 2008 ACS on STN
TI Antisense DNAs to cyclins and cyclin kinases for inhibition of proliferation of vascular smooth muscle cells

L39 ANSWER 40 OF 44 CAPLUS COPYRIGHT 2008 ACS on STN
TI Surface-modified nanoparticles and method of making and using them

L39 ANSWER 41 OF 44 CAPLUS COPYRIGHT 2008 ACS on STN
TI Ribozymes cleaving growth factor mRNAs for treatment of restenosis and cancers

L39 ANSWER 42 OF 44 CAPLUS COPYRIGHT 2008 ACS on STN
TI Method for treating diseases mediated by cellular proliferation in response to PDGF, EGF, FGF and VEGF

L39 ANSWER 43 OF 44 CAPLUS COPYRIGHT 2008 ACS on STN
TI Interdigitation-fusion liposomes containing arachidonic acid metabolites

L39 ANSWER 44 OF 44 CAPLUS COPYRIGHT 2008 ACS on STN
TI Inhibition of proliferation of vascular smooth muscle cells by antisense

oligonucleotides against cyclins and cyclin-dependent kinases

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FULL ESTIMATED COST                                    53.88     452.00

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)          SINCE FILE      TOTAL
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LAST RELOADED: Mar  7, 2008 (20080307/UP).

=> d l39 2 3 4 6 14 18 19 20 28 29 30 32 42 43 44 ti abs bib
YOU HAVE REQUESTED DATA FROM FILE 'CAPLUS' - CONTINUE? (Y)/N:y



L39  ANSWER 2 OF 44  CAPLUS  COPYRIGHT 2008 ACS on STN
TI   Oxidized lipids and uses thereof in the treatment of inflammatory diseases
     and disorders
AB   The invention provides synthetic oxidized lipids and methods using
     oxidized lipids for treating and preventing an inflammation associated with
     an endogenous oxidized lipid.
AN   2007:486401  CAPLUS <<LOGINID::20080311>>
DN   146:475683
TI   Oxidized lipids and uses thereof in the treatment of inflammatory diseases
     and disorders
IN   Harats, Dror; George, Jacob; Halperin, Gideon; Mendel, Itzhak
PA   Israel
SO   U.S. Pat. Appl. Publ., 87pp., Cont.-in-part of U.S. Ser. No. 567,543.
     CODEN: USXXCO
DT   Patent
LA   English
FAN.CNT 3
     PATENT NO.          KIND   DATE        APPLICATION NO.        DATE
     ---------------     ----   --------    --------------------   --------
PI   US 2007099868        A1    20070503    US 2006-528657         20060928 <--
     US 2003225035        A1    20031204    US 2003-445347         20030527 <--
     US 6838452           B2    20050104
     WO 2004106486        A2    20041209    WO 2004-IL453          20040527 <--
     WO 2004106486        A3    20050106
          W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH,
             CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD,
             GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC,
             LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI,
             NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY,
             TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
          RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM,
             AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK,
             EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE,
             SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE,
             SN, TD, TG
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PRAI US 2003-445347 A1 20030527 <--
WO 2004-IL453 W 20040527
US 2006-567543 A2 20060208
US 2000-252574P P 20001124 <--
WO 2001-IL101080 A2 20011122 <--
OS MARPAT 146:475683

L39 ANSWER 3 OF 44 CAPLUS COPYRIGHT 2008 ACS on STN
TI Use of lipid conjugates in the treatment of diseases
AB This invention provides lipid conjugates, i.e., compds. represented by the structure of the general formula [L-Z-Y]nX (L = lipid, phospholipid; Z = nothing, ethanolamine, serine, inositol, choline, glycerol; Y = nothing, spacer group ranging in length from 2 to 30 atoms; X ; monomer, dimer, oligomer, polymer, glycosaminoglycan; n = 1 to 1000; wherein any bond between L, Z, Y and X is either an amide or an esteric bond). Administration of these compds. comprises effective treatment of a subject afflicted with diseases involving the production of lipid mediators and/or impairment of glycosaminoglycan functioning. For example, CM-cellulose was conjugated to dipalmitoyl phosphatidylethanolamine (PE) to obtain a CMPE conjugate. The CMPE conjugate was effective in the treatment of obstructive respiratory disease, as demonstrated in asthma models. At a dose of 10 μM, CMPE inhibited quinea pig tracheal ring constriction induced by phospholipase (0.5 μ/mL) and endothelin-1 (100 nM) by 100% and 92%, resp. CMPE also reduced mortality of rats with TNBS-induced ulcerative colitis (9 out of 46 animals died compared to 27 of 46 in the control PBS-treated group).
AN 2005:1004345 CAPLUS <<LOGINID::20080311>>
DN 143:292563
TI Use of lipid conjugates in the treatment of diseases
IN Yedgar, Saul
PA Israel
SO U.S. Pat. Appl. Publ., 129 pp., Cont.-in-part of U. S. Ser. No. 756,765.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 12

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 2005203054	A1	20050915	US 2004-952496	20040929 <--
	US 2002049183	A1	20020425	US 2001-756765	20010110 <--
	US 7034006	B2	20060425		
	US 2006079485	A1	20060413	US 2005-220965	20050908 <--
	US 2006189568	A1	20060824	US 2005-220964	20050908 <--
	US 2006189569	A1	20060824	US 2005-220966	20050908 <--
	US 2006189570	A1	20060824	US 2005-220967	20050908 <--
	US 2006189571	A1	20060824	US 2005-220968	20050908 <--
	US 2007155700	A1	20070705	US 2006-475240	20060627 <--
	WO 2007029258	A2	20070315	WO 2006-IL1048	20060907
	WO 2007029258	A3	20070614		

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     US 2007078107        A1    20070405    US 2006-524519         20060921 <--
     US 2007117779        A1    20070524    US 2006-598812         20061114 <--
PRAI US 2000-174907P      P     20000110  <--
     US 2001-756765       A2    20010110  <--
     US 2000-174905P      P     20000110  <--
     US 2003-627981       A2    20030728  <--
     US 2004-790182       A2    20040302
     US 2004-952496       A2    20040929
     US 2004-989607       A2    20041117
     US 2005-220964       A     20050908
     US 2005-220965       A1    20050908
     US 2005-220967       A     20050908
OS   MARPAT 143:292563

L39  ANSWER 4 OF 44  CAPLUS  COPYRIGHT 2008 ACS on STN
TI   Methods and compounds for the treatment of vascular stenosis using a
     combination of N-phenyl-2-pyrimidine derivatives and PI3K inhibitors
AB   This invention features a method of treatment for vascular stenosis or
     restenosis using a combination of N-phenyl-2-pyrimidine derivs.
     such as imatinib mesylate and PI3K inhibitors, such as rapamycin.
AN   2004:1080793  CAPLUS <<LOGINID::20080311>>
DN   142:32971
TI   Methods and compounds for the treatment of vascular stenosis using a
     combination of N-phenyl-2-pyrimidine derivatives and PI3K inhibitors
IN   Sukhatme, Vikas P.
PA   Beth Israel Deaconess Medical Center, USA
SO   PCT Int. Appl., 48 pp.
     CODEN: PIXXD2
DT   Patent
LA   English
FAN.CNT 1
     PATENT NO.           KIND   DATE        APPLICATION NO.        DATE
     ---------------      ----   --------    -------------------    --------
PI   WO 2004108130        A1     20041216    WO 2004-US17273        20040601 <--
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             GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC,
             LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI,
             NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY,
             TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
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             AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK,
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             SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE,
             SN, TD, TG
     CA 2528032            A1     20041216    CA 2004-2528032        20040601 <--
     EP 1635815            A1     20060322    EP 2004-753981         20040601 <--
         R:  AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
             IE, SI, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK
     JP 2006526652         T      20061124    JP 2006-515065         20040601 <--
     US 2006240014         A1     20061026    US 2006-559057         20060530 <--
PRAI US 2003-475295P       P      20030603  <--
     WO 2004-US17273       W      20040601
RE.CNT  2     THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD
              ALL CITATIONS AVAILABLE IN THE RE FORMAT

L39  ANSWER 6 OF 44  CAPLUS  COPYRIGHT 2008 ACS on STN
TI   Method and system for systemic delivery of growth arresting, lipid-derived
     bioactive compounds
AB   A system and method for optimizing the systemic delivery of
     growth-arresting lipid-derived bioactive drugs or gene therapy agents to
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an animal or human in need of such agents utilizing nanoscale assembly systems, such as liposomes, resorbable and non-aggregating nanoparticle dispersions, metal or semiconductor nanoparticles, or polymeric materials such as dendrimers or hydrogels, each of which exhibit improved lipid solubility, cell permeability, an increased circulation half life and pharmacokinetic profile with improved tumor or vascular targeting.

AN 2004:965003 CAPLUS <<LOGINID::20080311>>

DN 141:400948

TI Method and system for systemic delivery of growth arresting, lipid-derived bioactive compounds

IN Kester, Mark; Stover, Thomas; Lowe, Tao; Adair, James; Kim, Young Shin

PA The Penn State Research Foundation, USA

SO PCT Int. Appl., 68 pp.
CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE	
PI	WO 2004096140	A2	20041111	WO 2004-US12783	20040426	<--
	WO 2004096140	A3	20050331			
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW					
	RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG					
	AU 2004233873	A1	20041111	AU 2004-233873	20040426	<--
	CA 2523413	A1	20041111	CA 2004-2523413	20040426	<--
	US 2005025820	A1	20050203	US 2004-835520	20040426	<--
	EP 1617808	A2	20060125	EP 2004-760381	20040426	<--
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK, HR					
	BR 2004009663	A	20060418	BR 2004-9663	20040426	<--
	CN 1812766	A	20060802	CN 2004-80017899	20040426	<--
	JP 2006524707	T	20061102	JP 2006-513321	20040426	<--
	IN 2005DN04874	A	20070928	IN 2005-DN4874	20051025	<--
PRAI	US 2003-465938P	P	20030425	<--		
	US 2003-465937P	P	20030428	<--		
	WO 2004-US12783	W	20040426			

L39 ANSWER 14 OF 44 CAPLUS COPYRIGHT 2008 ACS on STN

TI Drug delivery device with protective separating layer

AB The present invention relates to implantable medical devices for delivery of drugs to a patient. More particularly, the invention relates to a device having the drugs protected by a protective layer that prevents or retards processes that deactivate or degrade the active agents. Thus, a mixture of poly(lactide-co-glycolide) (PLGA) 7% by weight and a suitable organic solvent, such as DMSO, NMP, or DMAC 93% is prepared The mixture is loaded dropwise into holes in the stent, then the solvent is evaporated to begin formation of the barrier layer. A second barrier layer is laid over the first by the same method of filling polymer solution into the hole followed by solvent evaporation The process is continued until 5 individual layers have been laid down to form the barrier layer. A second mixture of a limus, such as sirolimus, 3% solid basis, and

dipalmitoylphosphatidylcholine 7% solid basis in DMSO is introduced into holes in the stent over the barrier layer. The solvent is evaporated to form a drug filled protective layer and the filling and evaporation procedure repeated until the hole is filled to about 75% of its total volume with drug in protective layer layered on top of the barrier layer.

AN 2003:281958 CAPLUS <<LOGINID::20080311>>

DN 138:292774

TI Drug delivery device with protective separating layer

IN Shanley, John F.; Parker, Theodore L.

PA USA

SO U.S. Pat. Appl. Publ., 17 pp., Cont.-in-part of U.S. Ser. No. 948,989.
CODEN: USXXCO

DT Patent

LA English

FAN.CNT 10

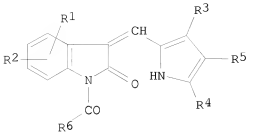
	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE	
PI	US 2003068355	A1	20030410	US 2002-253020	20020923	<--
	US 7208011	B2	20070424			
	US 2002082680	A1	20020627	US 2001-948989	20010907	<--
	US 7208010	B2	20070424			
	CA 2499475	A1	20040401	CA 2003-2499475	20030922	<--
	WO 2004026357	A1	20040401	WO 2003-US30125	20030922	<--
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW					
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG					
	AU 2003275229	A1	20040408	AU 2003-275229	20030922	<--
	EP 1551473	A1	20050713	EP 2003-759501	20030922	<--
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK					
	EP 1749544	A1	20070207	EP 2006-21147	20030922	<--
	R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LI, LU, MC, NL, PT, RO, SE, SI, SK, TR					
	US 2007219628	A1	20070920	US 2007-692770	20070328	<--
	AU 2007240255	A1	20080110	AU 2007-240255	20071212	
PRAI	US 2001-314259P	P	20010820	<--		
	US 2001-948989	A2	20010907	<--		
	US 2000-688092	A2	20001016	<--		
	US 2002-253020	A	20020923	<--		
	EP 2003-759501	A3	20030922	<--		
	WO 2003-US30125	W	20030922	<--		
	AU 2004-203857	A3	20040812			

RE.CNT 380 THERE ARE 380 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L39 ANSWER 18 OF 44 CAPLUS COPYRIGHT 2008 ACS on STN

TI Preparation of prodrugs of 3-(pyrrol-2-ylmethylidene)-2-indolinones and activity as modulators of protein kinases

GI



I

AB The present invention relates to pyrrole substituted 2-indolinone compds. (shown as I; e.g. 3-[1-(3,5-dimethyl-1H-pyrrol-2-yl)meth-(Z)-ylidene]-2-oxo-2,3-dihydroindole-1-carbonyl chloride) and their pharmaceutically acceptable salts which modulate the activity of protein kinases and therefore are expected to be useful in the prevention and treatment of protein kinase related cellular disorders such as cancer (no data). In I, R1 and R2 are independently H, halo, alkyl, alkylthio, nitro, trihalomethyl, hydroxy, hydroxyalkyl, alkoxy, cyano, aryl, heteroaryl, -C(O)R7 (R7 is alkyl, amino, hydroxy, alkoxy, aryl, heteroaryl, aryloxy, heteroaryloxy, heterocycle, and aminoalkylamino), -NR8R9, -NR8C(O)R9, -SO2R8, and -S(O)2NR8R9 (R8 and R9 are independently H, alkyl, aryl and heteroaryl, or R8 and R9 together with the N to which they are attached form a saturated heterocycloamino). R3 is H, alkyl, hydroxyalkyl, aminoalkyl, -C(O)R7, aryl, and heteroaryl; R4 is H, alkyl, -C(O)R7 aryl, and heteroaryl. R5 is H and -COR10 where R10 is alkyl, alkoxy, hydroxy, aryl, aryloxy, heteroaryl, heterocycle, alkylamino, dialkylamino, or -NR11R12 where R11 is H or alkyl, and R12 is aminoalkyl, hydroxyalkyl, acetylalkyl, cyanoalkyl, carboxyalkyl, alkoxycarbonylalkyl, heteroaralkyl, or heterocyclylalkyl wherein the alkyl chain in aminoalkyl, heteroaralkyl, heteroaralkyl, or heterocyclylalkyl is optionally substituted with one or two hydroxy group(s); or R4 and R5 together form - (CH2)4- or -(CH2)mCO(CH2)n- wherein n is 0 to 3, provided that n+m is 3. R6 is: (c) -OR13 wherein R13 is alkyl, trifluoromethyl, carboxyalkyl, aminoalkyl, phosphonooxyalkyl, sulfooxyalkyl, hydroxyalkyl, alkoxyalkyl, aryl, heteroaryl, heteroaralkyl, heterocyclyl, monosaccharides and heterocyclylalkyl wherein the alkyl chain in carboxyalkyl, aminoalkyl, phosphonooxyalkyl, sulfooxyalkyl, heteroaralkyl, heterocyclylalkyl, hydroxyalkyl, or alkoxyalkyl is optionally substituted with one or two hydroxy group(s) and further wherein one or two C atoms in said alkyl chain are optionally replaced by O, -NR14- (R14 is H or alkyl), -S-, or -SO2-; or. (d) -NR15R16 where are R15 and R16 are independently H, alkyl, carboxyalkyl, alkoxyalkyl, aminoalkyl, phosphonooxyalkyl, sulfooxyalkyl, hydroxyalkyl, aryl, heteroaryl, heteroaralkyl, and heterocyclylalkyl; wherein the alkyl chain in carboxyalkyl, aminoalkyl, phosphonooxyalkyl, heteroaralkyl, heterocyclylalkyl, hydroxyalkyl, or alkoxyalkyl is optionally substituted with one or two hydroxy group(s) and further wherein one or two C atoms in the alkyl chain are optionally replaced by O, -NR17- (R17 is H or alkyl), -S-, or -SO2-; or R15 and R16 together with the N atom to which they are attached form saturated or unsatd. heterocycloamino;. Although the methods of preparation are not claimed, >80 example prepns. are included, both of I and the unprotected version of I in which the C(O)R6 group has been replaced by H.

AN 2002:793619 CAPLUS <<LOGINID::20080311>>

DN 137:294870

TI Preparation of prodrugs of 3-(pyrrol-2-ylmethylidene)-2-indolinones and activity as modulators of protein kinases

IN Sun, Connie Li; Wei, Chung Chen; Tang, Peng Cho; Koenig, Marcel; Zhou, Yong; Vojkovsky, Tomas; Nemataila, Asaad S.

PA Sugen, Inc., USA
SO PCT Int. Appl., 194 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE	
PI	WO 2002081466	A1	20021017	WO 2002-US11001	20020409	<--
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW					
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG					
	AU 2002307183	A1	20021021	AU 2002-307183	20020409	<--
	US 2003100555	A1	20030529	US 2002-118321	20020409	<--
	US 6797725	B2	20040928			
	US 2004186161	A1	20040923	US 2004-816957	20040405	<--
	US 7053114	B2	20060530			
PRAI	US 2001-282630P	P	20010409	<--		
	US 2002-118321	A3	20020409	<--		
	WO 2002-US11001	W	20020409	<--		

OS MARPAT 137:294870
RE.CNT 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L39 ANSWER 19 OF 44 CAPLUS COPYRIGHT 2008 ACS on STN
TI Methods employing and compositions containing defined oxidized phospholipids for prevention and treatment of atherosclerosis
AB Novel synthetic forms of etherified oxidized phospholipids and methods of utilizing same for preventing and treating atherosclerosis and other related disorders, such as cardiovascular disease, cerebrovascular disease, peripheral vascular disease, stenosis, restenosis, etc., are provided. For example, an effective inhibition of late stage atherogenesis was observed in genetically predisposed (apoE-deficient) mice following protracted oral exposure to moderate doses (1 mg/mouse) of synthetic oxidized LDL components, hexadecyl-2-(5'-oxopentanyl)-sn-glycerophosphocholine (ALLE) and 1-hexadecanoyl-2-(5'-oxo)pentanoyl-sn-3-glycerophosphocholine (POVPC) (preparation given), compared to PBS-fed control mice. Induction of oral tolerance had no significant effect on other parameters measured, such as weight gain, total triglyceride or cholesterol blood levels. Surprisingly, it was observed that the inhibition of atherogenesis by these oxidized LDL analogs was accompanied by a significant reduction in VLDL cholesterol and triglycerides.
AN 2002:408469 CAPLUS <<LOGINID::20080311>>
DN 136:395962
TI Methods employing and compositions containing defined oxidized phospholipids for prevention and treatment of atherosclerosis
IN Harats, Dror; George, Jacob; Halperin, Gideon
PA Cardimmune Ltd., Israel; Vascular Biogenics Ltd.
SO PCT Int. Appl., 73 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 3

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE

```
PI   WO 2002041827        A2    20020530    WO 2001-IL1080         20011122 <--
     WO 2002041827        A3    20021010
     WO 2002041827        A9    20030530
          W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
              CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH,
              GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR,
              LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL,
              PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG,
              US, UZ, VN, YU, ZA, ZW
          RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AM, AZ, BY, KG,
              KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR,
              IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN,
              GQ, GW, ML, MR, NE, SN, TD, TG
     CA 2429817           A1    20020530    CA 2001-2429817        20011122 <--
     AU 2002018461        A     20020603    AU 2002-18461          20011122 <--
     EP 1341543           A2    20030910    EP 2001-997274         20011122 <--
          R:  AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
              IE, SI, LT, LV, FI, RO, MK, CY, AL, TR
     CN 1529605           A     20040915    CN 2001-822215         20011122 <--
     JP 2004537498        T     20041216    JP 2002-544008         20011122 <--
     MX 2003PA04517       A     20040326    MX 2003-PA4517         20030522 <--
     IN 2003CN00796       A     20050415    IN 2003-CN796          20030522 <--
     US 2003225035        A1    20031204    US 2003-445347         20030527 <--
     US 6838452           B2    20050104
     US 2004106677        A1    20040603    US 2003-718596         20031124 <--
     US 7186704           B2    20070306
     US 2005272813        A1    20051208    US 2005-183884         20050719 <--
     AU 2007200090        A1    20070201    AU 2007-200090         20070109 <--
PRAI US 2000-252574P      P     20001124  <--
     WO 2001-IL1080       W     20011122  <--
     US 2003-445347       A3    20030527  <--
     US 2003-718596       A3    20031124  <--
OS   MARPAT 136:395962

L39  ANSWER 20 OF 44  CAPLUS  COPYRIGHT 2008 ACS on STN
TI   Receptor antagonist-lipid conjugates and delivery vehicles containing same
AB   Vesicular drug delivery vehicles, such as liposomes, comprise a targeting
     ligand which comprises a non-biol., biomimetic antagonist to a receptor
     that is upregulated at a disease site, directly or indirectly chemical linked
     to a polar head group of a vesicle-forming lipid.  The non-biol.,
     biomimetic antagonist is an antagonist to a receptor upregulated in the
     vascular endothelium of inflammation, infection or tumor sites, selected
     from integrin receptors, prostate specific membrane antigen (PSMA)
     receptor, herceptin, Tie 1 and Tie 2 receptors, ICAM1, folate receptor,
     bFGF receptor, EGF receptor, VEGF receptor, PDGF receptor, etc.  The
     vesicle-forming lipid is selected from phospholipids, sterols,
     glycolipids, cationic lipids, sphingolipids, glycerolipids, hydrophilic
     polymer derivs. of these lipids, gemini surfactants, etc.  For example,
     liposomes were prepared containing lipid conjugates with a vitronectin receptor
     antagonist, (S)-7-[[N--(4-aminobutyl)-N-(benzimidazol-2-yl-
     methyl)]amino]carbonyl-4-methyl-3-oxo-2,3,4,5-tetrahydro-1H-1,4-
     benzodiazepine-2-acetic acid (preparation given) 0.5 mol%, DSPC 54.5 mol%, and
     cholesterol 45 mol%.  The liposomes were loaded with topotecan using ion
     gradient or polymer gradient loading/retaining techniques and administered
     to a patient diagnosed with ovarian cancer to inhibit growth of the
     cancerous tumor.  A dosing regimen was 1.5 mg/m2 of the topotecan
     liposomes given as a 30 min infusion over the course of 1-3 days in a week
     for 2 wk in a 21 day cycle, repeated for 4 cycles.
AN   2002:353239  CAPLUS <<LOGINID::20080311>>
DN   136:374827
TI   Receptor antagonist-lipid conjugates and delivery vehicles containing same
```

IN Ellens, Harma M.; Monck, Myrna A.; Yeh, Ping-Yang
PA Smithkline Beecham Corporation, USA
SO PCT Int. Appl., 44 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2002036073	A2	20020510	WO 2001-US46206	20011029 <--
	WO 2002036073	A3	20021205		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
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	AU 2002025878	A5	20020515	AU 2002-25878	20011029 <--
	EP 1341497	A2	20030910	EP 2001-992551	20011029 <--
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	JP 2004512345	T	20040422	JP 2002-538885	20011029 <--
	US 2004013720	A1	20040122	US 2003-415160	20030425 <--
PRAI	US 2000-245140P	P	20001102	<--	
	WO 2001-US46206	W	20011029	<--	

OS MARPAT 136:374827

L39 ANSWER 28 OF 44 CAPLUS COPYRIGHT 2008 ACS on STN
TI Preparation of vitronectin receptor antagonist pharmaceuticals
AB Compds. (Q)d-Ln-Ch (Q is a residue having a benzodiazepine-, benzodiazepinedione-, or dibenzotrihydroannulene-type moiety , d = 1-10, Ln is a linking group, Ch is a metal-bonding unit) were prepared for use in the diagnosis and treatment of cancer, methods of imaging tumors in a patient, and methods of treating cancer in a patient. The present invention also provides novel compds. useful for monitoring therapeutic angiogenesis treatment and destruction of new angiogenic vasculature. Thus, (S,S,S)-4-[N-[3-[3,6-diaza-10-[N-(benzimidazol-2-ylmethyl)-N-methylcarbamoyl]-5-(carboxymethyl)-4-oxobicyclo[5.4.0]undeca-1(7),8,10-trien-3-yl]propyl]carbamoyl]-4-[[4-carboxy-2-[2-[1,4,7,10-tetraaza-4,7,10-tris(carboxymethyl)cyclodecyl]acetylamino]butanoyl]amino]butanoic acid was prepared (claimed compound). Syntheses of radiopharmaceuticals, e.g., 99mTc(VnA)(tricine)(phosphine), where VnA represents the vitronectin receptor antagonist, are also described.
AN 2000:421115 CAPLUS <<LOGINID::20080311>>
DN 133:59101
TI Preparation of vitronectin receptor antagonist pharmaceuticals
IN Cheesman, Edward H.; Sworin, Michael; Rajopadhyem, Milind
PA Du Pont Pharmaceuticals Co., USA
SO PCT Int. Appl., 228 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 8

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2000035887	A2	20000622	WO 1999-US30311	19991217 <--
	WO 2000035887	A3	20001116		
	W: AL, AU, BR, CA, CN, CZ, EE, HU, IL, IN, JP, KR, LT, LV, MK, MX,				

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            NO, NZ, PL, RO, SG, SI, SK, TR, UA, VN, ZA, AM, AZ, BY, KG, KZ,
            MD, RU, TJ, TM
        RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL,
            PT, SE
    US 6322770          B1    20011127    US 1999-281207          19990330 <--
    US 2002015680       A1    20020207    US 1999-281209          19990330 <--
    US 6524553          B2    20030225
    US 6548663          B1    20030415    US 1999-281050          19990330 <--
    CA 2349333          A1    20000622    CA 1999-2349333         19991217 <--
    EP 1140864          A2    20011010    EP 1999-967441          19991217 <--
        R:  AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
            IE, SI, LT, LV, FI, RO
    TR 200101757        T2    20011221    TR 2001-1757            19991217 <--
    US 2003124120       A1    20030703    US 2002-269252          20021011 <--
    US 2003149262       A1    20030807    US 2002-306054          20021126 <--
PRAI US 1998-112831P    P     19981218  <--
    US 1998-80150P      P     19980331  <--
    US 1998-112715P     P     19981218  <--
    US 1998-112732P     P     19981218  <--
    US 1998-112829P     P     19981218  <--
    US 1999-281050      A3    19990330  <--
    US 1999-281209      A3    19990330  <--
    WO 1999-US30311     W     19991217  <--
OS  MARPAT 133:59101

L39 ANSWER 29 OF 44  CAPLUS  COPYRIGHT 2008 ACS on STN
TI  Preparation of vitronectin receptor antagonist pharmaceuticals
AB  Compds. (Q)d-Ln-Ch (Q is a residue having a quinolone-type moiety , d =
    1-10, Ln is a linking group, Ch is a metal-bonding unit) were prepared for
    use in the diagnosis and treatment of cancer, methods of imaging tumors in
    a patient, and methods of treating cancer in a patient.  The present
    invention also provides novel compds. useful for monitoring therapeutic
    angiogenesis treatment and destruction of new angiogenic vasculature.
    Thus, [3-[1-[3-[3-[N-[3-[2-[N-(L-Asp-L-Asp)-3-
    aminopropoxy]ethoxy]ethoxy]propyl]carbamoyl]propanoylamino]propyl]-7-
    [(imidazol-2-ylamino)methyl]-4-oxo(3-hydroquinolyl)carbonylamino]-2-
    [[(2,4,6-trimethylphenyl)sulfonyl]amino]propanoic acid DOTA conjugate was
    prepared (claimed compound).  Syntheses of radiopharmaceuticals, e.g.,
    99mTc(VnA)(tricine)(phosphine), where VnA represents the vitronectin
    receptor antagonist, are also described.
AN  2000:420994  CAPLUS <<LOGINID::20080311>>
DN  133:59099
TI  Preparation of vitronectin receptor antagonist pharmaceuticals
IN  Harris, Thomas David; Rajodadhye, Milind
PA  Du Pont Pharmaceuticals Company, USA
SO  PCT Int. Appl., 300 pp.
    CODEN: PIXXD2
DT  Patent
LA  English
FAN.CNT 8
    PATENT NO.          KIND   DATE        APPLICATION NO.         DATE
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PI  WO 2000035492       A2     20000622    WO 1999-US30315         19991217 <--
    WO 2000035492       A3     20010118
        W:  AL, AU, BR, CA, CN, CZ, EE, HU, IL, IN, JP, KR, LT, LV, MK, MX,
            NO, NZ, PL, RO, SG, SI, SK, TR, UA, VN, ZA, AM, AZ, BY, KG, KZ,
            MD, RU, TJ, TM
        RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL,
            PT, SE
    US 6322770          B1     20011127    US 1999-281207          19990330 <--
    US 2002015680       A1     20020207    US 1999-281209          19990330 <--
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US 6524553	B2	20030225			
US 6548663	B1	20030415	US 1999-281050	19990330	<--
CA 2349501	A1	20000622	CA 1999-2349501	19991217	<--
EP 1140204	A2	20011010	EP 1999-967443	19991217	<--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO					
BR 9917079	A	20011030	BR 1999-17079	19991217	<--
JP 2002532440	T	20021002	JP 2000-587811	19991217	<--
AU 766822	B2	20031023	AU 2000-23716	19991217	<--
NZ 511677	A	20031031	NZ 1999-511677	19991217	<--
ZA 2001003675	A	20020607	ZA 2001-3675	20010507	<--
IN 2001MN00576	A	20050304	IN 2001-MN576	20010522	<--
MX 2001PA06151	A	20020311	MX 2001-PA6151	20010615	<--
US 2003124120	A1	20030703	US 2002-269252	20021011	<--
US 2003149262	A1	20030807	US 2002-306054	20021126	<--
PRAI US 1998-112732P	P	19981218	<--		
US 1998-80150P	P	19980331	<--		
US 1998-112715P	P	19981218	<--		
US 1998-112829P	P	19981218	<--		
US 1998-112831P	P	19981218	<--		
US 1999-281050	A3	19990330	<--		
US 1999-281209	A3	19990330	<--		
WO 1999-US30315	W	19991217	<--		

OS MARPAT 133:59099

L39 ANSWER 30 OF 44 CAPLUS COPYRIGHT 2008 ACS on STN

TI Preparation of vitronectin receptor antagonist pharmaceuticals

AB Compds. (Q)d-Ln-Ch (Q is a residue having an indazole-type moiety , d = 1-10, Ln is a linking group, Ch is a metal-bonding unit) were prepared for use in the diagnosis and treatment of cancer, methods of imaging tumors in a patient, and methods of treating cancer in a patient. The present invention also provides novel compds. useful for monitoring therapeutic angiogenesis treatment and destruction of new angiogenic vasculature. Thus, 2-[[[4-[4-[[[3-[2-[2-[3-[[6-[[1-aza-2-(2-sulfophenyl)vinyl]amino](3-pyridyl)]carbonylamino]propoxy]ethoxy]ethoxy]propyl]amino]sulfonyl]phenyl]phenyl]sulfonyl]amino]-3-[[1-[3-(indazole-2-ylamino)propyl](1H-indazol-5-yl)]carbonylamino]propanoic acid was prepared (claimed compound). Syntheses of radiopharmaceticals, e.g., 99mTc(VnA)(tricine)(phosphine), where VnA represents the vitronectin receptor antagonist, are also described.

AN 2000:420991 CAPLUS <<LOGINID::20080311>>

DN 133:59098

TI Preparation of vitronectin receptor antagonist pharmaceuticals

IN Rajopadhye, Milind; Harris, Thomas David; Cheesman, Edward H.

PA Du Pont Pharmaceuticals Company, USA

SO PCT Int. Appl., 362 pp.

CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 8

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE	
PI WO 2000035488	A2	20000622	WO 1999-US30312	19991217	<--
WO 2000035488	A3	20001109			
W: AL, AU, BR, CA, CN, CZ, EE, HU, IL, IN, JP, KR, LT, LV, MK, MX, NO, NZ, PL, RO, SG, SI, SK, TR, UA, VN, ZA, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM					
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE					
US 6322770	B1	20011127	US 1999-281207	19990330	<--
US 2002015680	A1	20020207	US 1999-281209	19990330	<--
US 6524553	B2	20030225			

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     US 6548663          B1    20030415    US 1999-281050        19990330 <--
     CA 2346935          A1    20000622    CA 1999-2346935       19991217 <--
     AU 2000023715       A     20000703    AU 2000-23715         19991217 <--
     EP 1140203          A2    20011010    EP 1999-967442        19991217 <--
     EP 1140203          B1    20070523
          R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
             IE, SI, LT, LV, FI, RO, CY
     TR 200101775        T2    20020722    TR 2001-1775          19991217 <--
     AT 362772           T     20070615    AT 1999-967442        19991217 <--
     ES 2288040          T3    20071216    ES 1999-967442        19991217 <--
     US 2003124120       A1    20030703    US 2002-269252        20021011 <--
     US 2003149262       A1    20030807    US 2002-306054        20021126 <--
PRAI US 1998-112829P     P     19981218  <--
     US 1998-80150P      P     19980331  <--
     US 1998-112715P     P     19981218  <--
     US 1998-112732P     P     19981218  <--
     US 1998-112831P     P     19981218  <--
     US 1999-281050      A3    19990330  <--
     US 1999-281209      A3    19990330  <--
     WO 1999-US30312     W     19991217  <--
OS   MARPAT 133:59098

L39  ANSWER 32 OF 44  CAPLUS  COPYRIGHT 2008 ACS on STN
TI   Agent for gene therapy of tumors and neurodegenerative, cardiovascular,
     and autoimmune diseases
AB   A method for local/regional gene therapy of tumors (especially liver
metastases)
     and of neurodegenerative, cardiovascular, and autoimmune diseases
     comprises combined application of liposomes/plasmid DNA complexes having
     different compns., quantities, and concns.  The pharmaceutical agent
     employed comprises ≥1 genetic material which are nonencapsulated or
     encapsulated in PEG, immuno-, immuno/PEG, or cationic, optionally
     polymer-modified liposomes; lyophilized or degradable starch particles
     and/or gelatin and/or polymer nanoparticles; and a contrast agent containing
     I, Gd, magnetite, or F.  The genetic material preferably constitutes a
     suicide gene such as herpes simplex virus thymidine kinase (HSV-tk) gene,
     deaminase gene, or a cytokine gene coding for IL-2, IL-4, IL-6, IL-10,
     IL-12, or IL-15, and is enclosed in multilamellar liposomes comprising an
     amphiphile, a steroid, and an anionic lipid.  Thus, phosphatidylcholine-
     cholesterol-PEG liposomes containing suicide gene pUT 649, which encodes
     HSV-tk, were injected together with a drug carrier embolization system
     into the common hepatic artery of rats which had been inoculated with
     CC531 carcinoma cells 10 days previously.  Beginning 5 days later, the
     rats were treated with ganciclovir (100 mg/kg/day i.p.) for 14 days.  The
     rats showed a decrease in liver metastases after 30 days owing to
     conversion of ganciclovir by HSV-tk to a nucleotide-like compound which was
     incorporated into the DNA of dividing liver cells, causing cessation of
     DNA synthesis.
AN   1999:404862  CAPLUS <<LOGINID::20080311>>
DN   131:39728
TI   Agent for gene therapy of tumors and neurodegenerative, cardiovascular,
     and autoimmune diseases
IN   Reszka, Regina; Berndt, Antje
PA   Max-Delbrueck-Centrum fuer Molekulare Medizin, Germany
SO   PCT Int. Appl., 28 pp.
     CODEN: PIXXD2
DT   Patent
LA   German
FAN.CNT 1
     PATENT NO.          KIND   DATE        APPLICATION NO.       DATE
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PI   WO 9930741          A2    19990624    WO 1998-DE3763         19981214 <--
     WO 9930741          A3    19990819
          W:  JP, US
          RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL,
              PT, SE
     DE 19859526         A1    19990819    DE 1998-19859526       19981214 <--
     EP 1037670          A2    20000927    EP 1998-966568         19981214 <--
     EP 1037670          B1    20031105
          R:  AT, BE, CH, DE, DK, FR, GB, IT, LI, NL, SE, FI
     JP 2002508337       T     20020319    JP 2000-538719         19981214 <--
     AT 253379           T     20031115    AT 1998-966568         19981214 <--
PRAI DE 1997-19756309    A     19971212  <--
     WO 1998-DE3763      W     19981214  <--

L39  ANSWER 42 OF 44  CAPLUS  COPYRIGHT 2008 ACS on STN
TI   Method for treating diseases mediated by cellular proliferation in
     response to PDGF, EGF, FGF and VEGF
AB   There is disclosed a method for: (1) inhibiting new blood vessel formation
     that is useful for treating or preventing progression of diabetic
     retinopathy, cavernous hemangiomas, Kaposi's sarcoma, tumors composed of
     endothelial-like cells, and growth of solid tumors by preventing their
     development of a new blood supply; (2) suppressing development of kidney
     diseases due to cytokine induced proliferation of mesangial cells and/or
     glomerular epithelial cells that is useful for treating or preventing
     progression of diabetic glomerulosclerosis and other glomerulonephritides
     of various types and etiologies; (3) preventing joint destruction
     accompanying rheumatoid arthritis due to proliferation of synovial cells;
     (4) suppressing manifestations of psoriasis due to proliferation of
     keratinocytes and accumulation of inflammatory cells; (5) suppressing
     accelerated atherogenesis involved in restenosis of coronary
     vessels or other arterial vessels following angioplasty; (6) suppressing
     atherogenesis, coronary artery disease and other vasculopathies due to
     atherogenesis; and (7) suppressing tumor growth via paracrine or autocrine
     mediated responses to PDGF, FGF, EGF, or VEGF.  This is useful for
     treating or preventing progression of tumors such as breast cancer
     stimulated through overexpression of her-2-neu receptor, wherein the
     inventive method comprises administering a compound that inhibits signal
     transduction through cellular accumulation of phosphatidic acid having
     predominantly linoleate and a C22 alkyl or alkenyl in the sn-2 position or
     a vinyl ether alkenyl group in the sn-1 position.
AN   1995:804532  CAPLUS <<LOGINID::20080311>>
DN   123:276007
TI   Method for treating diseases mediated by cellular proliferation in
     response to PDGF, EGF, FGF and VEGF
IN   Brown, Paul A.; Bursten, Stuart L.; Rice, Glenn C.; Singer, Jack W.
PA   Cell Therapeutics, Inc., USA
SO   PCT Int. Appl., 58 pp.
     CODEN: PIXXD2
DT   Patent
LA   English
FAN.CNT 1
     PATENT NO.          KIND   DATE        APPLICATION NO.        DATE
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PI   WO 9519171          A1     19950720    WO 1995-US520          19950113 <--
          W:  AU, CA, JP
          RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE
     CA 2192470          A1     19950720    CA 1995-2192470        19950113 <--
     AU 9518313          A      19950801    AU 1995-18313          19950113 <--
     EP 739203           A1     19961030    EP 1995-910088         19950113 <--
          R:  AT, DE, ES, FR, GB, IE, IT
     US 5795898          A      19980818    US 1995-485325         19950607 <--
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     US 5859018           A     19990112    US 1995-485322          19950607 <--
     US 5929081           A     19990727    US 1995-485320          19950607 <--
PRAI US 1994-181947       A     19940114  <--
     WO 1995-US520        W     19950113  <--
OS   MARPAT 123:276007

L39  ANSWER 43 OF 44  CAPLUS  COPYRIGHT 2008 ACS on STN
TI   Interdigitation-fusion liposomes containing arachidonic acid metabolites
AB   An interdigitation-fusion liposome comprises an arachidonic acid
     metabolite, e.g. a prostaglandin, a lipid bilayer comprising a lipid, and
     an aqueous compartment comprising a release-inhibiting buffer.  The liposomal
     formulations can be used to treat animals, particularly humans, for
     diseases, disorders or conditions which can be ameliorated by
     prostaglandins, e.g. cell activation/adhesion disorders and inflammatory
     disorders.  A solution of 1mg/mL PGE1 was combined with a solution of
     dipalmitoylphosphatidylcholine at a weight ratio of PGE1:lipid = 1:20, then
     the solvent evaporated  The dried mixture was then rehydrated with an aqueous
solution
     of 50mM citrate buffer to form a suspension of multilamellar liposomes.
AN   1995:780419  CAPLUS <<LOGINID::20080311>>
DN   123:179480
TI   Interdigitation-fusion liposomes containing arachidonic acid metabolites
IN   Janoff, Andrew S.; Minchey, Sharma R.
PA   Liposome Co., Inc., USA
SO   PCT Int. Appl., 34 pp.
     CODEN: PIXXD2
DT   Patent
LA   English
FAN.CNT 5
     PATENT NO.           KIND   DATE        APPLICATION NO.         DATE
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PI   WO 9513797           A1     19950526    WO 1994-US13063         19941115 <--
         W:  AU, CA, FI, JP, KR, NO
         RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE
     CA 2175350           A1     19950526    CA 1994-2175350         19941115 <--
     AU 9510555           A      19950606    AU 1995-10555           19941115 <--
     AU 681469            B2     19970828
     EP 729352            A1     19960904    EP 1995-901236          19941115 <--
     EP 729352            B1     19990203
         R:  AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE
     JP 09505302          T      19970527    JP 1995-514532          19941115 <--
     JP 3798016           B2     20060719
     AT 176397            T      19990215    AT 1995-901236          19941115 <--
     ES 2126868           T3     19990401    ES 1995-901236          19941115 <--
     NO 9601949           A      19960513    NO 1996-1949            19960513 <--
     NO 312808            B1     20020708
     FI 9602080           A      19960515    FI 1996-2080            19960515 <--
PRAI US 1993-153176       A      19931116  <--
     WO 1994-US13063      W      19941115  <--

L39  ANSWER 44 OF 44  CAPLUS  COPYRIGHT 2008 ACS on STN
TI   Inhibition of proliferation of vascular smooth muscle cells by antisense
     oligonucleotides against cyclins and cyclin-dependent kinases
AB   This invention encompasses a method for inhibiting vascular cellular
     activity of cells associated with vascular lesion formation in mammals which
     involves administering an effective dosage of at least one antisense
     sequence to at least one gene expressing a cyclin or a cyclin-dependent
     kinase.  More particularly, the invention involves administering antisense
     sequences which inhibit the expression of cyclin A, B1, B2, C, D1, D2, D3,
     E or cyclin X(p46) and cyclin-dependent kinases cdc2, cdk2, cdk4 or cdk5.
     It is preferable to use 2 antisense sequences each from a different cyclin
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or cyclin-dependent kinase. The cyclin or cyclin-dependent kinase dosage is preferably administered in combination with proliferating cell nuclear antigen (PCNA). Antisense methods and compns. directed toward inhibiting the expression of growth factors such as TGF-β1, TGF, bFGF, PDGF are also provided. The antisense sequences are incorporated into liposomes, particularly liposomes containing HVJ (hemagglutinating virus of Japan) and are directly administered intraluminally, intramurally, or periadventiously. The methods of this invention are useful in treating a broad spectrum of vascular lesions such as lesions in the carotid, femural, and renal arteries, and particularly lesions resulting from renal dialysis fistulas. The invention is particularly useful in treating vascular lesions associated with coronary artery angioplasty.

AN 1995:380320 CAPLUS <<LOGINID::20080311>>

DN 122:151381

TI Inhibition of proliferation of vascular smooth muscle cells by antisense oligonucleotides against cyclins and cyclin-dependent kinases

IN Dzau, Victor J.

PA Board of Trustees of the Leland Stanford Junior University, USA

SO PCT Int. Appl., 76 pp.
CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 4

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE	
PI	WO 9426888	A1	19941124	WO 1994-US5566	19940518	<--
	W: CA, JP					
	RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE					
	US 5821234	A	19981013	US 1993-110294	19930820	<--
	EP 701609	A1	19960320	EP 1994-919161	19940518	<--
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE					
	JP 09507381	T	19970729	JP 1994-525809	19940518	<--
PRAI	US 1993-63980	A	19930519	<--		
	US 1993-110294	A	19930820	<--		
	US 1992-944882	B2	19920910	<--		
	WO 1994-US5566	W	19940518	<--		